result in loss of user privileges and other penalties.

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FILE 'HOME' ENTERED AT 16:33:13 ON 24 JAN 2007

=>
<------User Break------->

=> file .meeting
'EVENTLINE' IS NOT A VALID FILE NAME
Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files
that are available. If you have requested multiple files, you can
specify a corrected file name or you can enter "IGNORE" to continue
accessing the remaining file names entered.
ENTER A FILE NAME OR (IGNORE):ignore
'MEDICONF' IS NOT A VALID FILE NAME
Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files
that are available. If you have requested multiple files, you can
specify a corrected file name or you can enter "IGNORE" to continue
accessing the remaining file names entered.
ENTER A FILE NAME OR (IGNORE):ignore
COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION
FULL ESTIMATED COST 3.78 3.78

FILE 'AGRICOLA' ENTERED AT 16:44:05 ON 24 JAN 2007

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=> mercury and (autism or autistic) and (antigen or antibody)
L1 0 FILE AGRICOLA
L2 0 FILE BIOTECHNO
L3 0 FILE CONFSCI
L4 0 FILE HEALSAFE
L5 0 FILE IMSDRUGCONF
L6 3 FILE LIFESCI
L7 1 FILE PASCAL

TOTAL FOR ALL FILES
L8 4 MERCURY AND (AUTISM OR AUTISTIC) AND (ANTIGEN OR ANTIBODY)

=> dup rem

ENTER L# LIST OR (END):l8
DUPLICATE IS NOT AVAILABLE IN 'IMSDRUGCONF'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE
PROCESSING COMPLETED FOR L8
L9 3 DUP REM L8 (1 DUPLICATE REMOVED)

=> d l9 ibib abs total

L9 ANSWER 1 OF 3 LIFESCI COPYRIGHT 2007 CSA on STN DUPLICATE 1
ACCESSION NUMBER: 2006:126670 LIFESCI
TITLE: Assessment of metallothionein and antibodies to metallothionein in normal and autistic children having exposure to vaccine-derived thimerosal
AUTHOR: Singh, Vijendra K.; Hanson, Jeff
CORPORATE SOURCE: Department of Biology, Utah State University, Logan, UT, USA; E-mail: singhvk@cc.usu.edu
SOURCE: Pediatric Allergy and Immunology, (20060600) vol. 17, no. 4, pp. 291-296. Figures, 3..
ISSN: 0905-6157.
DOCUMENT TYPE: Journal
FILE SEGMENT: X
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Allergic autoimmune reaction after exposure to heavy metals such as mercury may play a causal role in autism, a developmental disorder of the central nervous system. As metallothionein (MT) is the primary metal-detoxifying protein in the body, we conducted a study of the MT protein and antibodies to metallothionein (anti-MT) in normal and autistic children whose exposure to mercury was only from thimerosal-containing vaccines. Laboratory analysis by immunoassays revealed that the serum level of MT did not significantly differ between normal and autistic children. Furthermore, autistic children harboured normal levels of anti-MT, including antibodies to isoform MT-I (anti-MT-I) and MT-II (anti-MT-II), without any significant difference between normal and autistic children. Our findings indicate that because autistic children have a normal profile of MT and anti-MT, the mercury-induced autoimmunity to MT may not be implicated in the pathogenesis of autism.

L9 ANSWER 2 OF 3 LIFESCI COPYRIGHT 2007 CSA on STN
ACCESSION NUMBER: 2005:56113 LIFESCI
TITLE: Detection of Antinuclear and Antilaminin Antibodies in Autistic Children Who Received Thimerosal-Containing Vaccines
AUTHOR: Singh, V.K.; Rivas, W.H.
CORPORATE SOURCE: Biotechnology Center Building, Utah State University, UMC 4700, Logan, UT 84322 (USA); E-mail: singhvk@cc.usu.edu
SOURCE: Journal of Biomedical Science [J. Biomed. Sci.], (20041000) vol. 11, no. 5, pp. 607-610.
ISSN: 1021-7770.
DOCUMENT TYPE: Journal
FILE SEGMENT: X
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Autism, a neurodevelopmental disorder, may involve autoimmune pathogenesis. Since mercury is potentially a risk factor for autoimmunity, we conducted a study of mercury-induced antinuclear and antilaminin antibodies in autistic and normal children who had been pre-administered with thimerosal-containing vaccines. Laboratory analysis by different immunoassays showed that the serum level of these two autoimmune markers did not significantly differ between autistic and normal children. This finding suggests that the mercury as in thimerosal-containing vaccines is likely not

related to autoimmune phenomenon in autism.

L9 ANSWER 3 OF 3 LIFESCI COPYRIGHT 2007 CSA on STN
ACCESSION NUMBER: 2004:108019 LIFESCI
TITLE: Infections, toxic chemicals and dietary peptides binding to lymphocyte receptors and tissue enzymes are major instigators of autoimmunity in autism
AUTHOR: Vojdani, A.; Pangborn, J.B.; Vojdani, E.; Cooper, E.L.
CORPORATE SOURCE: 8693 Wilshire Blvd., Ste. 200, Beverly Hills, CA 90211, USA; E-mail: DrAri@msn.com
SOURCE: International Journal of Immunopathology and Pharmacology [Int. J. Immunopathol. Pharmacol.], (20031200) vol. 16, no. 3, pp. 189-199.
ISSN: 0394-6320.
DOCUMENT TYPE: Journal
FILE SEGMENT: F
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Similar to many complex autoimmune diseases, genetic and environmental factors including diet, infection and xenobiotics play a critical role in the development of autism. In this study, we postulated that infectious agent antigens such as streptokinase, dietary peptides (gliadin and casein) and ethyl mercury (xenobiotic) bind to different lymphocyte receptors and tissue enzyme (DPP IV or CD26). We assessed this hypothesis first by measuring IgG, IgM and IgA antibodies against CD26, CD69, streptokinase (SK), gliadin and casein peptides and against ethyl mercury bound to human serum albumin in patients with autism. A significant percentage of children with autism developed anti-SK, anti-gliadin and casein peptides and anti-ethyl mercury antibodies, concomitant with the appearance of anti-CD26 and anti-CD69 autoantibodies. These antibodies are synthesized as a result of SK, gliadin, casein and ethyl mercury binding to CD26 and CD69, indicating that they are specific. Immune absorption demonstrated that only specific antigens, like CD26, were capable of significantly reducing serum anti-CD26 levels. However, for direct demonstration of SK, gliadin, casein and ethyl mercury to CD26 or CD69, microtiter wells were coated with CD26 or CD69 alone or in combination with SK, gliadin, casein or ethyl mercury and then reacted with enzyme labeled rabbit anti-CD26 or anti-CD69. Adding these molecules to CD26 or CD69 resulted in 28-86% inhibition of CD26 or CD69 binding to anti-CD26 or anti-CD69 antibodies. The highest % binding of these antigens or peptides to CD26 or CD69 was attributed to SK and the lowest to casein peptides. We, therefore, propose that bacterial antigens (SK), dietary peptides (gliadin, casein) and Thimerosal (ethyl mercury ) in individuals with pre-disposing HLA molecules, bind to CD26 or CD69 and induce antibodies against these molecules. In conclusion, this study is apparently the first to demonstrate that dietary peptides, bacterial toxins and xenobiotics bind to lymphocyte receptors and/or tissue enzymes, resulting in autoimmune reaction in children with autism.

=> file .chemistry
COST IN U.S. DOLLARS

| | SINCE FILE ENTRY | TOTAL SESSION |
|---|---|---|
| FULL ESTIMATED COST | 14.20 | 17.98 |

FILE 'CAPLUS' ENTERED AT 16:45:51 ON 24 JAN 2007
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FILE 'COMPENDEX' ENTERED AT 16:45:51 ON 24 JAN 2007


FILE 'ANABSTR' ENTERED AT 16:45:51 ON 24 JAN 2007


FILE 'CERAB' ENTERED AT 16:45:51 ON 24 JAN 2007


FILE 'METADEX' ENTERED AT 16:45:51 ON 24 JAN 2007


FILE 'USPATFULL' ENTERED AT 16:45:51 ON 24 JAN 2007


=> (CD13, CD26, transglutaminase, DPPI, dipeptidyl peptidase IV, secretin, gastrin, motilin) and (autism or autistic) and (antigen or antibody)
L10 0 FILE CAPLUS
L11 0 FILE BIOTECHNO
L12 0 FILE COMPENDEX
L13 0 FILE ANABSTR
L14 0 FILE CERAB
L15 0 FILE METADEX
L16 0 FILE USPATFULL

TOTAL FOR ALL FILES
L17 0 (CD13, CD26, TRANSGLUTAMINASE, DPPI, DIPEPTIDYL PEPTIDASE IV, SECRETIN, GASTRIN, MOTILIN) AND (AUTISM OR AUTISTIC) AND (ANTIGEN OR ANTIBODY)

=> (autism or autistic) and (antigen or antibody) and (CD13 or CD26 or transglutaminase or DPPI or dipeptidyl peptidase IV or secretin or gastrin or motilin)
L18 17 FILE CAPLUS
L19 0 FILE BIOTECHNO
L20 1 FILE COMPENDEX
L21 0 FILE ANABSTR
L22 0 FILE CERAB
L23 0 FILE METADEX
L24 403 FILE USPATFULL

TOTAL FOR ALL FILES
L25 421 (AUTISM OR AUTISTIC) AND (ANTIGEN OR ANTIBODY) AND (CD13 OR CD26 OR TRANSGLUTAMINASE OR DPPI OR DIPEPTIDYL PEPTIDASE IV OR SECRETIN OR GASTRIN OR MOTILIN)

=> dup rem
ENTER L# LIST OR (END):18
18 IS NOT VALID HERE
The L-number entered has not been defined in this session, or it has been deleted. To see the L-numbers currently defined in this session, enter DISPLAY HISTORY at an arrow prompt (=>).

=> dup rem
ENTER L# LIST OR (END):l18
PROCESSING COMPLETED FOR L18
L26 17 DUP REM L18 (0 DUPLICATES REMOVED)

=> d l26 ibib abs total

L26 ANSWER 1 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2006:1205478 CAPLUS
DOCUMENT NUMBER: 145:503524
TITLE: Identification of diagnostic biomarkers for autism by multiplatform analysis of blood cell immunophenotype and serum protein and metabolite content
INVENTOR(S): Amaral, David G.; Corbett, Blythe A.
PATENT ASSIGNEE(S): The Regents of the University of California, USA
SOURCE: PCT Int. Appl., 82pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2006121952 | A2 | 20061116 | WO 2006-US17581 | 20060505 |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW | | | | |
| RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | | |
| US 2007003922 | A1 | 20070104 | US 2006-381976 | 20060505 |
| PRIORITY APPLN. INFO.: | | | US 2005-678865P P | 20050505 |

AB The present invention is directed to methods for diagnosing neurodevelopmental disorders, including autism, by employing a multiplatform anal. of blood cell immunophenotype and serum polypeptide and metabolite content. The present invention provides methods of identifying biomarkers indicative of the presence of a neurodevelopmental disorder, including an autism spectrum disorder, in an individual, using cytometry and mass spectrometry. The invention further provides methods of using the identified biomarkers to diagnose the presence of a neurodevelopmental disorder, including an autism spectrum disorder.

L26 ANSWER 2 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:962041 CAPLUS
DOCUMENT NUMBER: 143:242034
TITLE: DPP-IV inhibitors for neurodegenerative and cognitive disorders
INVENTOR(S): Hughes, Thomas Edward
PATENT ASSIGNEE(S): Novartis A.-G., Switz.; Novartis Pharma G.m.b.H.
SOURCE: PCT Int. Appl., 44 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2005079795 | A2 | 20050901 | WO 2005-EP1729 | 20050218 |
| WO 2005079795 | A3 | 20051110 | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, | | | | |

GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, SM
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT,
RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML,
MR, NE, SN, TD, TG

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|---|---|---|---|---|
| AU 2005215136 | A1 | 20050901 | AU 2005-215136 | 20050218 |
| CA 2555399 | A1 | 20050901 | CA 2005-2555399 | 20050218 |
| EP 1732550 | A2 | 20061220 | EP 2005-707520 | 20050218 |

R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
IS, IT, LI, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR

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|---|---|---|
| PRIORITY APPLN. INFO.: | US 2004-546229P | P 20040220 |
| | US 2004-607902P | P 20040908 |
| | WO 2005-EP1729 | W 20050218 |

AB The invention relates to the use of a dipeptidyl peptidase IV inhibitor (DPP-IV inhibitor) or a pharmaceutically acceptable salt thereof for the prevention, delay of progression or the treatment of neurodegenerative disorders, cognitive disorders and for improving memory (both short term and long term) and learning ability.

L26 ANSWER 3 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN

| | |
|---|---|
| ACCESSION NUMBER: | 2005:281768 CAPLUS |
| DOCUMENT NUMBER: | 142:354292 |
| TITLE: | Use of proline-specific endoproteases to hydrolyse proline-rich peptides at acid pH in food processing |
| INVENTOR(S): | Edens, Luppo; Van Der Hoeven, Robertus Antonius Mijndert; De Roos, Andre Leonardus; Harvey, Melissa |
| PATENT ASSIGNEE(S): | DSM IP Assets B.V., Neth. |
| SOURCE: | PCT Int. Appl., 56 pp.<br>CODEN: PIXXD2 |
| DOCUMENT TYPE: | Patent |
| LANGUAGE: | English |
| FAMILY ACC. NUM. COUNT: | 1 |

PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2005027953 | A2 | 20050331 | WO 2004-EP10782 | 20040923 |
| WO 2005027953 | A3 | 20050616 | | |

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE,
SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
SN, TD, TG

| | | | | |
|---|---|---|---|---|
| EP 1663298 | A2 | 20060607 | EP 2004-765616 | 20040923 |

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK

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|---|---|---|
| PRIORITY APPLN. INFO.: | EP 2003-78012 | A 20030923 |
| | EP 2003-78496 | A 20031106 |
| | WO 2004-EP10782 | W 20040923 |

AB A method of processing protein-rich food products, such as milk, to eliminate proline-rich peptides uses prolyl oligopeptidases to hydrolyze such peptides on the C-terminal side of the proline. This is particularly useful in treating milk products, such as caseins, to eliminate

casomorphins. The enzymes have an acid pH optimum and are members of the subtilisin family, but can use these proline-rich substrates that are resistant to other serine proteinases. The enzymes also accept a limited size range of peptides (4-40 amino acids) as substrates. Removal of these peptides in food processing is useful in the prevention of psychiatric and autoimmune disorders. The enzymes may be administered orally to a patient as needed because of their acid pH optima. A proline endopeptidase of Aspergillus nigers was used in combination with the com. subtilisin Alcalase to degrade caseins. Alcalase alone generated significant quantities of β-casomorphin from β-caseins. When used in combination with the proline endopeptidase, the yield of β-casomorphin was lowered by ≥2000-fold.

L26 ANSWER 4 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:698214 CAPLUS
DOCUMENT NUMBER: 143:171341
TITLE: Methods for detecting infections, toxic chemicals and dietary peptides binding to lymphocyte receptors and tissue enzymes as instigators of autoimmunity in autism
INVENTOR(S): Vojdani, Aristo
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 89 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| US 2005170333 | A1 | 20050804 | US 2004-770712 | 20040203 |

PRIORITY APPLN. INFO.: US 2004-770712 20040203

AB The present invention provides methods for diagnosis and following up a prognosis of children with autism before and after treatment with different modalities administered by their clinicians, confirming the involvement of infectious agents, dietary proteins, and toxic chems. in development of autism. In particular, methods for detecting infections, toxic chems. and dietary peptides binding to lymphocyte receptors and tissue enzymes as instigators of autoimmunity in autism are described. The method utilizes detection of increased amts. of antibodies against an antigen based on infectious agent, toxic chems., or dietary proteins. Another method utilizes detection of antibodies to a self-tissue or peptide.

L26 ANSWER 5 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:496904 CAPLUS
DOCUMENT NUMBER: 141:205490
TITLE: Heat shock protein and gliadin peptide promote development of peptidase antibodies in children with autism and patients with autoimmune disease
AUTHOR(S): Vojdani, Aristo; Bazargan, Mohsen; Vojdani, Elroy; Samadi, John; Nourian, Alen A.; Eghbalieh, Navid; Cooper, Edwin L.
CORPORATE SOURCE: Laboratory of Comparative Neuroimmunology, Department of Neurobiology, David Geffen School of Medicine, University of California, Los Angeles, CA, 90095, USA
SOURCE: Clinical and Diagnostic Laboratory Immunology (2004), 11(3), 515-524
CODEN: CDIMEN; ISSN: 1071-412X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Searching for a mechanism underlying autoimmunity in autism, the authors postulated that gliadin peptides, heat shock protein 60 (HSP-60), and streptokinase (SK) bind to different peptidases resulting in autoantibody production against these components. The authors assessed this hypothesis in patients with autism and in those with mixed connective tissue diseases. Associated with anti-gliadin and anti-HSP antibodies, children with autism and patients with autoimmune disease developed anti-dipeptidylpeptidase I (DPP I), anti-dipeptidylpeptidase IV (DPP IV [or CD26]), and anti-aminopeptidase N (CD13) autoantibodies. A percentage of autoimmune and autistic sera were associated with elevated IgG, IgM, or IgA antibodies against 3 peptidases, gliadin, and HSP-60. These antibodies are specific, since immune absorption demonstrated that only specific antigens (e.g., DPP IV absorption of anti-DPP IV), reduced IgG, IgM, and IgA antibody levels. For direct demonstration of SK, HSP-60, and gliadin peptide binding to DPP IV, microtiter wells coated with DPP IV were reacted with SK, HSP-60, and gliadin. They were then reacted with anti-DPP IV or anti-SK, anti-HSP, and antigliadin antibodies. Adding SK, HSP-60, and gliadin peptides to DPP IV resulted in 27-43% inhibition of the DPP IV-anti-DPP IV reaction, but DPP IV-pos. peptides caused 18-20% enhancement of antigen-antibody reactions. The authors propose that (1) superantigens (e.g., SK and HSP-60) and dietary proteins (e.g., gliadin peptides) in individuals with predisposing HLA mols. bind to aminopeptidases and (2) they induce autoantibodies to peptides and tissue antigens. Dysfunctional membrane peptidases and autoantibody production may result in neuroimmune dysregulation and autoimmunity.

REFERENCE COUNT: 64 THERE ARE 64 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L26 ANSWER 6 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:185188 CAPLUS
DOCUMENT NUMBER: 141:52075
TITLE: Secretin: Hypothalamic Distribution and Hypothesized Neuroregulatory Role in Autism
AUTHOR(S): Welch, M. G.; Keune, J. D.; Welch-Horan, T. B.; Anwar, N.; Anwar, M.; Ludwig, R. J.; Ruggiero, D. A.
CORPORATE SOURCE: Laboratories of Childhood Regulatory Disorders and Behavorial Neuroanatomy, College of Physicians and Surgeons, Division of Neuroscience, NYSPI, Columbia University, New York, NY, USA
SOURCE: Cellular and Molecular Neurobiology (2004), 24(2), 219-241
CODEN: CMNEDI; ISSN: 0272-4340
PUBLISHER: Kluwer Academic/Plenum Publishers
DOCUMENT TYPE: Journal
LANGUAGE: English

AB This study aims (1) to determine whether secretin is synthesized centrally, specifically by the HPA axis and (2) to discuss, on the basis of the findings in this and previous studies, secretin's possible neuroregulatory role in autism. An immunocytochem. technique with single-cell resolution was performed in 12 age/weight-matched male rats pretreated with stereotaxic microinjection of colchicine (0.6 μg/kg) or vehicle into the lateral ventricle. Following 2-day survival, rats were anesthetized and perfused for immunocytochem. Brain segments were blocked and alternate frozen 30-μm sections incubated in rabbit antibodies against secretin, vasoactive intestinal peptide, glucagon, or pituitary-adenylate-cyclase-activating peptide. Adjacent sections were processed for Nissl stain. Preadsorption studies were performed with members of the secretin peptide family to demonstrate primary antibody specificity. Specificity of secretin immunoreactivity (ir) was verified by clear-cut preadsorption control data and relatively high concns. and distinct topog.

localization of secretin ir to paraventricular/supraoptic and intercalated hypothalamic nuclei. Secretin levels were upregulated by colchicine, an exemplar of homeostatic stressors, as compared with low constitutive expression in untreated rats. This study provides the first direct immunocytochem. demonstration of secretinergic immunoreactivity in the forebrain and offers evidence that the hypothalamus, like the gut, is capable of synthesizing secretin. Secretin's dual expression by gut and brain secretin cells, as well as its overlapping central distribution with other stress-adaptation neurohormones, especially oxytocin, indicates that it is stress-sensitive. A neuroregulatory relationship between the peripheral and central stress response systems is suggested, as is a dual role for secretin in conditioning both of those stress-adaptation systems. Colchicine-induced upregulation of secretin indicates that secretin may be synthesized on demand in response to stress, a possible mechanism of action that may underlie secretin's role in autism.

REFERENCE COUNT: 136 THERE ARE 136 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L26 ANSWER 7 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2003:397078 CAPLUS

DOCUMENT NUMBER: 138:397218

TITLE: Multi-parameter high throughput screening assays (MPHTS) for identifying therapeutic compounds for treatment of neuropsychiatric and neurodegenerative disorders

INVENTOR(S): Altar, Anthony C.; Brockman, Jeffrey A.; Evans, David; Hook, Derek; Klimczak, Leszek; Laeng, Pascal; Palfreyman, Michael; Rajan, Prithi

PATENT ASSIGNEE(S): Psychiatric Genomics, Inc., USA

SOURCE: PCT Int. Appl., 103 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | | DATE |
|---|---|---|---|---|---|
| WO 2003042654 | A2 | 20030522 | WO 2002-US31106 | | 20020927 |
| WO 2003042654 | A9 | 20030807 | | | |
| WO 2003042654 | A3 | 20040603 | | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW | | | | | |
| RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | | |
| US 2003096264 | A1 | 20030522 | US 2002-175523 | | 20020618 |
| AU 2002368043 | A1 | 20040123 | AU 2002-368043 | | 20020618 |
| AU 2002327791 | A1 | 20030526 | AU 2002-327791 | | 20020927 |
| PRIORITY APPLN. INFO.: | | | US 2001-333047P | P | 20011114 |
| | | | US 2002-349936P | P | 20020118 |
| | | | US 2002-361834P | P | 20020304 |
| | | | US 2002-175523 | A | 20020618 |
| | | | US 2001-299151P | P | 20010618 |
| | | | US 2001-317828P | P | 20010907 |
| | | | US 2001-325150P | P | 20010925 |

WO 2002-US19457 W 20020618
WO 2002-US31106 W 20020927

AB The present invention relates to screening methods and assays that are referred to herein as multi-parameter high throughput screening (MPHTS) assays. These methods pertain to the combination of data generated from gene expression profiling coupled with methods for the systematic anal. and/or employment of such data. Such methods comprise steps of: identifying a plurality of disease signature genes and identifying a plurality of drug signature genes, followed by obtaining a score value for each of these genes that is a function of each gene's differential expression in the disease signature compared to its expression in the drug signature. Once scored, disease signature and drug signature genes having the highest score(s) may then ben selected as efficacy genes. Large nos. of candidate compds may be screened in vitro to identify ones that are particularly suitable and promising as novel therapeutic agents. These MPHTS assays are useful for identifying candidate pharmaceutical compds. In particular, the screening methods of this invention may be used to identify compds. that have potential therapeutic benefits for the treatment of neuropsychiatric and neurodegenerative disorders, including schizophrenia, bipolar affective disorder (BAD), autism, and Alzheimer's disease to name a few.

L26 ANSWER 8 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:396350 CAPLUS
DOCUMENT NUMBER: 138:396234
TITLE: Multi-parameter high throughput screening assays (MPHTS) for screening of therapeutic candidates for neuropsychiatric and neurodegenerative disorders
INVENTOR(S): Altar, C. Anthony; Brockman, Jeffrey A.; Evans, David; Hook, Derek; Klimczak, Leszek J.; Laeng, Pascal; Palfreyman, Michael; Rajan, Prithi
PATENT ASSIGNEE(S): Psychiatric Genomics, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 39 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| US 2003096264 | A1 | 20030522 | US 2002-175523 | 20020618 |
| WO 2004005882 | A2 | 20040115 | WO 2002-US19457 | 20020618 |
| WO 2004005882 | A3 | 20050127 | | |

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, EC, EE, ES, FI, GB, GD, GE, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

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|---|---|---|---|---|
| AU 2002368043 | A1 | 20040123 | AU 2002-368043 | 20020618 |
| WO 2003042654 | A2 | 20030522 | WO 2002-US31106 | 20020927 |
| WO 2003042654 | A9 | 20030807 | | |
| WO 2003042654 | A3 | 20040603 | | |

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

| | | | | | |
|---|---|---|---|---|---|
| AU 2002327791 | A1 | 20030526 | AU 2002-327791 | | 20020927 |
| US 2005181433 | A1 | 20050818 | US 2005-99266 | | 20050404 |
| PRIORITY APPLN. INFO.: | | | US 2001-299151P | P | 20010618 |
| | | | US 2001-317828P | P | 20010907 |
| | | | US 2001-325150P | P | 20010925 |
| | | | US 2001-333047P | P | 20011114 |
| | | | US 2002-349936P | P | 20020118 |
| | | | US 2002-361834P | P | 20020304 |
| | | | US 2002-175523 | A | 20020618 |
| | | | WO 2002-US19457 | W | 20020618 |
| | | | WO 2002-US31106 | W | 20020927 |

AB The present invention relates to screening methods and assays that are referred to herein as multi-parameter high throughput screening (MPHTS) assays. These MPHTS assays are useful for identifying candidate pharmaceutical compds. In particular, the screening methods of this invention may be used to identify compds. that have potential therapeutic benefits for the treatment of neuropsychiatric and neurodegenerative disorders, including schizophrenia, bipolar affective disorder (BAD), autism and Alzheimer's disease to name a few.

L26 ANSWER 9 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN

| | |
|---|---|
| ACCESSION NUMBER: | 2003:605851 CAPLUS |
| DOCUMENT NUMBER: | 140:71310 |
| TITLE: | Secretin activates visceral brain regions in the rat including areas abnormal in autism |
| AUTHOR(S): | Welch, Martha G.; Keune, Jason D.; Welch-Horan, T. Bramwell; Anwar, Nargis; Anwar, Muhammad; Ruggiero, David A. |
| CORPORATE SOURCE: | Division of Neuroscience, Laboratories of Childhood Regulatory Disorders and Behavioral Neuroanatomy, Riverside Drive, NYSPI, Columbia University College of Physicians and Surgeons, Department of Psychiatry, Columbia University College of Physicians and Surgeons, New York, NY, USA |
| SOURCE: | Cellular and Molecular Neurobiology (2003), 23(4/5), 817-837<br>CODEN: CMNEDI; ISSN: 0272-4340 |
| PUBLISHER: | Kluwer Academic/Plenum Publishers |
| DOCUMENT TYPE: | Journal |
| LANGUAGE: | English |

AB The aim of this study was to determine whether central networks are involved in the presumptive behavioral and autonomic regulatory actions of secretin, a gut hormone that has been reported to have ameliorative effects in autistic children. Central neural responses monitored by regional c-fos gene expression were examined in response to intracerebroventricular secretin injection in awake, freely-moving Sprague-Dawley rats. Tissue sections were incubated in an antibody to the c-fos gene product, Fos, and processed immunohistochem. Qual. differences in Fos immunoreactivity in stress adaptation and visceral representation areas of the brain were observed between secretin- and vehicle-infused age-matched pairs. Secretin-activated regions include the area postrema, dorsal motor nucleus, medial region of the nucleus of the solitary tract and its relay station in the lateral tegmentum, locus ceruleus, ventral periaqueductal gray, periventricular thalamic nucleus, paraventricular hypothalamus magnocellularis, medial and central amygdala, lateral septal complex as well as ependymal and subependymal nuclei lining the third ventricle. Specific areas of the cerebral cortex were heavily labeled in secretin-treated rats, as compared to controls: the medial bank of the anterior prefrontal cortex, orbitofrontal cortex, the piriform cortex,

and the anterior olfactory nucleus. Secretin attenuated Fos immunoreactivity in the dorsal periaqueductal gray, intralaminar thalamus, medial parvicellular compartment of the hypothalamus, supraoptic nucleus of the hypothalamus, lateral amygdala, motor cortex, and the somatosensory and association areas of the parietal cortex. Secretin alters the activity of structures involved in behavioral conditioning of stress adaptation and visceral reflex reactions. This study predicts a possible cellular mechanism, activation of third ventricular ependymal and subependymal cells, as well as central regulatory actions of secretin. The physiol. effects of secretin on behavioral, endocrine, autonomic and sensory neuronal activation patterns, together, contribute to central c-fos activation. Secretin alters the activity of structures involved in behavioral conditioning of stress adaptation and visceral reflex reactions. This study predicts a possible cellular mechanism, activation of third ventricular ependymal and subependymal cells, and central regulatory actions of secretin. The physiol. effects of secretin on behavioral, endocrine, autonomic and sensory neuronal activation patterns, together, contribute to central c-fos activation. These findings mandate further investigation of secretin as a brain/gut stress regulatory hormone.

REFERENCE COUNT: 123 THERE ARE 123 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L26 ANSWER 10 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:306157 CAPLUS
DOCUMENT NUMBER: 139:34798
TITLE: Does the MMR vaccine and secretin or its receptor share an antigenic epitope?
AUTHOR(S): Mehta, Bijal K.; Munir, Kerim M.
CORPORATE SOURCE: Memorial University of Newfoundland, St. John's, NF, Can.
SOURCE: Medical Hypotheses (2003), 60(5), 650-653
CODEN: MEHYDY; ISSN: 0306-9877
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review and discussion. In a subgroup of children with autism -spectrum like conditions symptoms seem to appear as a regression (in normal development). It has been postulated that the onset of such autistic symptoms may involve an autoimmune response against the central nervous system and that the antigenic determinant could possibly be gastrointestinal in origin. It has been suggested that the presence of the measles virus and autistic enterocolitis demonstrates the possibility that the MMR triple vaccine may be mediating the inflammation with possible production of antibodies against the virus containing vaccine. Such an antibody may share antigenic determinant to mols. found in the gut. The authors propose that this may be secretin or its receptor, found in the gut as well as in the central nervous system. The antibody response to the gut may also conceivably occur in the brain at a critical time in development. The modulation of development by secretin may be a static event possibly occurring at a specific time in early childhood development and if it involves an autoimmune response then a disruption in development may result. These hypothesized events can only occur if the MMR vaccine shares antigenic determinants that resemble secretin or any of its receptor types.

REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L26 ANSWER 11 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:44082 CAPLUS
DOCUMENT NUMBER: 140:216004
TITLE: Infections, toxic chemicals and dietary peptides

binding to lymphocyte receptors and tissue enzymes are major instigators of autoimmunity in autism

AUTHOR(S): Vojdani, A.; Pangborn, J. B.; Vojdani, E.; Cooper, E. L.

CORPORATE SOURCE: Laboratory of Comparative Neuroimmunology, Department of Neurobiology, David Geffen School of Medicine at UCLA, University of California, Los Angeles, Los Angeles, CA, 90095, USA

SOURCE: International Journal of Immunopathology and Pharmacology (2003), 16(3), 189-199
CODEN: IJIPE4; ISSN: 0394-6320

PUBLISHER: Biolife s.a.s.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Similar to many complex autoimmune diseases, genetic and environmental factors including diet, infection and xenobiotics play a critical role in the development of autism. In this study, we postulated that infectious agent antigens such as streptokinase, dietary peptides (gliadin and casein) and Et mercury (xenobiotic) bind to different lymphocyte receptors and tissue enzyme (DPP IV or CD26 ). We assessed this hypothesis first by measuring IgG, IgM and IgA antibodies against CD26, CD69, streptokinase (SK), gliadin and casein peptides and against Et mercury bound to human serum albumin in patients with autism. A significant percentage of children with autism developed anti-SK, anti-gliadin and casein peptides and anti-Et mercury antibodies, concomitant with the appearance of anti-CD26 and anti-CD69 autoantibodies. These antibodies are synthesized as a result of SK, gliadin, casein and Et mercury binding to CD26 and CD69, indicating that they are specific. Immune absorption demonstrated that only specific antigens, like CD26, were capable of significantly reducing serum anti-CD26 levels. However, for direct demonstration of SK, gliadin, casein and Et mercury to CD26 or CD69, microtiter wells were coated with CD26 or CD69 alone or in combination with SK, gliadin, casein or Et mercury and then reacted with enzyme labeled rabbit anti-CD26 or anti-CD69. Adding these mols. to CD26 or CD69 resulted in 28-86% inhibition of CD26 or CD69 binding to anti-CD26 or anti-CD69 antibodies. The highest % binding of these antigens or peptides to CD26 or CD69 was attributed to SK and the lowest to casein peptides. We, therefore, propose that bacterial antigens (SK), dietary peptides (gliadin, casein) and Thimerosat (Et mercury) in individuals with pre-disposing HLA mols.; bind to CD26 or CD69 and induce antibodies against these mols. In conclusion, this study is apparently the first to demonstrate that dietary peptides, bacterial toxins and xenobiotics bind to lymphocyte receptors and/or tissue enzymes, resulting in autoimmune reaction in children with autism.

REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L26 ANSWER 12 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2002:906554 CAPLUS

DOCUMENT NUMBER: 138:1044

TITLE: G protein-coupled receptor (GPCR) microarrays for determination of GPCR gene expression profiles and uses in drug and toxin screening and diagnostics

INVENTOR(S): Thirstrup, Kenneth; Madsen, Lars Siim; Jensen, Jens Bitsch; Hummel, Rene; Jensen, Bo Skaaning

PATENT ASSIGNEE(S): Azign Bioscience A/s, Den.

SOURCE: PCT Int. Appl., 43 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2002095065 | A2 | 20021128 | WO 2002-DK337 | 20020521 |
| WO 2002095065 | A3 | 20040325 | | |
| W: | AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW | | | |
| RW: | GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | |
| EP 1423530 | A2 | 20040602 | EP 2002-724150 | 20020521 |
| R: | AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR | | | |
| US 2004171008 | A1 | 20040902 | US 2003-477399 | 20031112 |
| PRIORITY APPLN. INFO.: | | | DK 2001-802 A | 20010518 |
| | | | WO 2002-DK337 W | 20020521 |

AB The invention provides G protein-coupled receptor (GPCR) arrays, kits comprising GPCR arrays and methods to produce such GPCR arrays. GPCR arrays are useful in the determination of GPCR expression profiles in biol. materials and also in the identification of therapeutic, prophylactic and/or toxic agents involved in the response of the GPCR expression. The invention relates to an GPCR array comprising a multiplicity of individual GPCR polynucleotide spots stably associated with a surface of a solid support, wherein an individual GPCR polynucleotide spot comprises an GPCR polynucleotide composition comprising a non-conserved region of an GPCR polynucleotide family member, the spots representing at least two different regions of an GPCR polynucleotide member of a family. The invention also relates to a set of primers specific for nonconserved regions of GPCR polynucleotide family members, wherein the set of primers are used in the method for the production of an array according to the invention. In still a further aspect, the invention relates to a diagnostic method to determine the differences of GPCR expression profiles between two different biol. materials.

L26 ANSWER 13 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:276155 CAPLUS
DOCUMENT NUMBER: 136:305198
TITLE: Sequences of a novel human secretin receptor-like G protein-coupled receptor sequence homolog and uses in diagnosis, therapy and drug screening
INVENTOR(S): Liou, Jiing-Ren
PATENT ASSIGNEE(S): Bayer Aktiengesellschaft, Germany
SOURCE: PCT Int. Appl., 122 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2002029052 | A2 | 20020411 | WO 2001-EP11515 | 20011005 |
| WO 2002029052 | A3 | 20021227 | | |
| W: | AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, | | | |

LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL,
PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG,
US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

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|---|---|---|---|---|
| AU 2002010522 | A5 | 20020415 | AU 2002-10522 | 20011005 |
| EP 1326975 | A2 | 20030716 | EP 2001-978398 | 20011005 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR | | | | |
| US 2004048273 | A1 | 20040311 | US 2003-398454 | 20031008 |
| PRIORITY APPLN. INFO.: | | | US 2000-238125P | P 20001006 |
| | | | US 2001-261756P | P 20010117 |
| | | | WO 2001-EP11515 | W 20011005 |

AB The invention provides protein and cDNA sequences of a novel human secretin receptor-like G protein-coupled receptor sequence homolog. The invention also provides reagents and methods of regulating a human secretin receptor-like G protein-coupled receptor sequence homolog. Reagents which regulate human secretin receptor receptor-like GPCR and reagents which bind to human secretin receptor-like GPCR gene products can play a role in preventing, ameliorating, or correcting dysfunctions or diseases including, but not limited to, cardiovascular disorders, urinary incontinence, benign prostate hyperplasia, obesity and diseases related to obesity, cancer, diabetes, osteoporosis, anxiety, depression, hypertension, migraine, compulsive disorders, schizophrenia, autism, neurodegenerative disorders, such as Alzheimer's disease, Parkinsonism, and Huntington's chorea, and cancer chemotherapy-induced vomiting.

L26 ANSWER 14 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:276154 CAPLUS
DOCUMENT NUMBER: 136:305197
TITLE: Sequences of a novel human secretin receptor-like G protein-coupled receptor sequence homolog and uses in diagnosis, therapy and drug screening
INVENTOR(S): Liou, Jiing-Ren
PATENT ASSIGNEE(S): Bayer Aktiengesellschaft, Germany
SOURCE: PCT Int. Appl., 113 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2002029051 | A2 | 20020411 | WO 2001-EP11443 | 20011004 |
| WO 2002029051 | A3 | 20030320 | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | | |
| RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| AU 2002018205 | A5 | 20020415 | AU 2002-18205 | 20011004 |
| EP 1326977 | A2 | 20030716 | EP 2001-986319 | 20011004 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR | | | | |
| US 2004058350 | A1 | 20040325 | US 2003-398455 | 20030923 |
| PRIORITY APPLN. INFO.: | | | US 2000-238043P | P 20001006 |

WO 2001-EP11443 W 20011004

AB The invention provides protein and cDNA sequences of a novel human secretin receptor-like G protein-coupled receptor sequence homolog. The invention also provides reagents and methods of regulating a human secretin receptor-like G protein-coupled receptor sequence homolog. Reagents which regulate human secretin receptor receptor-like GPCR and reagents which bind to human secretin receptor-like GPCR gene products can play a role in preventing, ameliorating, or correcting dysfunctions or diseases including, but not limited to, cardiovascular disorders, urinary incontinence, benign prostate hyperplasia, obesity and diseases related to obesity, cancer, diabetes, osteoporosis, anxiety, depression, hypertension, migraine, compulsive disorders, schizophrenia, autism, neurodegenerative disorders, such as Alzheimer's disease, Parkinsonism, and Huntington's chorea, and cancer chemotherapy-induced vomiting.

L26 ANSWER 15 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:276153 CAPLUS
DOCUMENT NUMBER: 136:305196
TITLE: Sequences of a novel human secretin receptor-like G protein-coupled receptor sequence homolog and uses in diagnosis, therapy and drug screening
INVENTOR(S): Liou, Jiing-Ren
PATENT ASSIGNEE(S): Bayer Aktiengesellschaft, Germany
SOURCE: PCT Int. Appl., 133 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | | DATE |
|---|---|---|---|---|---|
| WO 2002029050 | A2 | 20020411 | WO 2001-EP11442 | | 20011004 |
| WO 2002029050 | A3 | 20030522 | | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW | | | | | |
| RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | | |
| AU 2002012309 | A5 | 20020415 | AU 2002-12309 | | 20011004 |
| EP 1328637 | A2 | 20030723 | EP 2001-980476 | | 20011004 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR | | | | | |
| US 2004096847 | A1 | 20040520 | US 2003-398448 | | 20030404 |
| PRIORITY APPLN. INFO.: | | | US 2000-238045P | P | 20001006 |
| | | | US 2001-315958P | P | 20010831 |
| | | | WO 2001-EP11442 | W | 20011004 |

AB The invention provides protein and cDNA sequences of a novel human secretin receptor-like G protein-coupled receptor sequence homolog. The invention also provides reagents and methods of regulating a human secretin receptor-like G protein-coupled receptor sequence homolog. Reagents which regulate human secretin receptor receptor-like GPCR and reagents which bind to human secretin receptor-like GPCR gene products can play a role in preventing, ameliorating, or correcting dysfunctions or diseases including, but not limited to, cardiovascular disorders, urinary incontinence, benign prostate hyperplasia, obesity and diseases related to obesity, cancer,

diabetes, osteoporosis, anxiety, depression, hypertension, migraine, compulsive disorders, schizophrenia, autism, neurodegenerative disorders, such as Alzheimer's disease, Parkinsonism, and Huntington's chorea, and cancer chemotherapy-induced vomiting.

L26 ANSWER 16 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:276025 CAPLUS
DOCUMENT NUMBER: 136:304110
TITLE: Regulation of human secretin receptor-like GPCR
INVENTOR(S): Kossida, Sophia
PATENT ASSIGNEE(S): Bayer Aktiengesellschaft, Germany
SOURCE: PCT Int. Appl., 125 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2002028898 | A2 | 20020411 | WO 2001-EP11439 | 20011004 |
| WO 2002028898 | A3 | 20030605 | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW | | | | |
| RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| AU 2002012308 | A5 | 20020415 | AU 2002-12308 | 20011004 |
| EP 1332215 | A2 | 20030806 | EP 2001-980475 | 20011004 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR | | | | |
| US 2004024184 | A1 | 20040205 | US 2003-398458 | 20030717 |

PRIORITY APPLN. INFO.: US 2000-238126P P 20001006
WO 2001-EP11439 W 20011004

AB Reagents which regulate human secretin receptor-like GPCR and reagents which bind to human secretin-like GPCR gene products can play a role in preventing, ameliorating, or correcting dysfunctions or diseases including, but not limited to, obesity and diseases related to obesity, cancer, diabetes, osteoporosis, anxiety, depression, hypertension, migraine, compulsive disorders, schizophrenia, autism, neurodegenerative disorders, such as Alzheimer's disease, Parkinsonism, and Huntington's chorea, and cancer chemotherapy-induced vomiting.

L26 ANSWER 17 OF 17 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:895643 CAPLUS
DOCUMENT NUMBER: 138:168045
TITLE: Effects of neonatal 6-hydroxydopamine lesion on the gene expression profile in young adult rats
AUTHOR(S): Masuo, Yoshinori; Ishido, Masami; Morita, Masatoshi; Oka, Syuichi
CORPORATE SOURCE: International Patent Organism Depositary, National Institute of Advanced Industrial Science and Technology (AIST), Tsukuba, Ibaraki, 305-8566, Japan
SOURCE: Neuroscience Letters (2002), 335(2), 124-128
CODEN: NELED5; ISSN: 0304-3940
PUBLISHER: Elsevier Science Ireland Ltd.
DOCUMENT TYPE: Journal

LANGUAGE: English
AB Patients with pervasive developmental disorders, including autism , and attention-deficit hyperactivity disorder show behavioral hyperactivity during childhood. We investigated the effects of a neonatal 6-hydroxydopamine lesion on multiple gene expression in the rat striatum and midbrain. Spontaneous motor activity was significantly increased at 4-5 wk of age. The animals were sacrificed, and the striatum and midbrain were subjected to gene expression profiling using a membrane array with 1176 kinds of cDNAs. Alterations were found in several classes of gene expression, depending on the brain region. Enhanced expression of the glutamate transporter gene was found in the striatum. Expression of the dopamine receptor D4 gene and dopamine transporter gene was also increased in the midbrain. These results suggest that 6-hydroxydopamine-treated rats may partly mimic human hyperkinesia not only in behavior but also in gene expression.
REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> (autism or autistic) and (antigen or antibody) and (enkephelin or substance P or somatostatin or serotonin or serotonin receptor)
L27 12 FILE CAPLUS
L28 1 FILE BIOTECHNO
L29 0 FILE COMPENDEX
L30 0 FILE ANABSTR
L31 0 FILE CERAB
L32 0 FILE METADEX
L33 527 FILE USPATFULL

TOTAL FOR ALL FILES
L34 540 (AUTISM OR AUTISTIC) AND (ANTIGEN OR ANTIBODY) AND (ENKEPHELIN OR SUBSTANCE P OR SOMATOSTATIN OR SEROTONIN OR SEROTONIN RECEPTOR)

=> dup rem
ENTER L# LIST OR (END):127
PROCESSING COMPLETED FOR L27
L35 12 DUP REM L27 (0 DUPLICATES REMOVED)

=> d l35 ibib abs total

L35 ANSWER 1 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2006:463638 CAPLUS
DOCUMENT NUMBER: 145:354047
TITLE: A Family Based Linkage Analysis of HLA and 5-HTTLPR Gene Polymorphisms in Sardinian Children with Autism Spectrum Disorder
AUTHOR(S): Guerini, Franca R.; Manca, Salvatorica; Sotgiu, Stefano; Tremolada, Sara; Zanzottera, Milena; Agliardi, Cristina; Zanetta, Lorenzo; Saresella, Marina; Mancuso, Roberta; De Silvestri, Annalisa; Fois, Maria Laura; Arru, Giannina; Ferrante, Pasquale
CORPORATE SOURCE: Don C. Gnocchi Foundation IRCCS, Laboratory of Molecular Medicine and Biotechnologies, Milan, Italy
SOURCE: Human Immunology (2006), 67(1-2), 108-117
CODEN: HUIMDQ; ISSN: 0198-8859
PUBLISHER: Elsevier Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Autism spectrum disorders (ASD) are characterized by a broad range in clin. presentation. Although a definite genetic cause has not yet been fully demonstrated, family based studies suggest that a multigenic pattern may be responsible for susceptibility, but most results are conflicting and have yet to be replicated. The purpose of this

investigation was to analyze the linkage of the human leukocyte antigen (HLA) and the human serotonin transporter coding (5-HTTLPR) genes with ASD in a group of 37 families of Sardinian ethnicity in insular Italy. In 50% of these families, ASD is linked to HLA, and in the other 50% it is linked to 5-HTTLPR polymorphic genes; in other words, linkage to one or the other was evident in all cases. Despite a very homogenous genetic pattern being generally reported for Sardinians, the linkage observed with HLA and 5-HTTLPR genetic regions indicated a statistically defined heterogeneity (p = 0.002). No allelic HLA or 5-HTTLPR polymorphisms were specifically associated with ASD, suggesting these loci as markers of other genes mapped in their close proximity that may be more directly involved and thus may merit further anal. studies.

REFERENCE COUNT: 64 THERE ARE 64 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L35 ANSWER 2 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:729611 CAPLUS
DOCUMENT NUMBER: 143:206465
TITLE: Therapeutic and carrier molecules
INVENTOR(S): Ferrante, Antonio; Rathjen, Deborah Ann
PATENT ASSIGNEE(S): Peplin Biolipids Pty Ltd, Australia
SOURCE: PCT Int. Appl., 180 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2005073164 | A1 | 20050811 | WO 2005-AU98 | 20050128 |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW | | | | |
| RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| AU 2005209331 | A1 | 20050811 | AU 2005-209331 | 20050128 |
| CA 2554735 | A1 | 20050811 | CA 2005-2554735 | 20050128 |
| EP 1718602 | A1 | 20061108 | EP 2005-700130 | 20050128 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK, IS | | | | |

PRIORITY APPLN. INFO.: US 2004-540604P P 20040130
WO 2005-AU98 W 20050128

OTHER SOURCE(S): MARPAT 143:206465

AB The present invention relates generally to compds. comprising a hydrocarbon chain portion and more particular to compds. comprising chemical derivatizations of the hydrocarbon chain which are useful therapeutic and prophylactic mols. The present invention further provides compds. where the hydrocarbon chain portion is a carrier mol. for functional groups, moieties or agents. The present invention can include naturally including polyunsatd. fatty acids as well as synthetic, modified or derivatized polyunsatd. fatty acids. Furthermore. these polyunsatd. fatty acids can be conjugated to amino acids, peptides or proteins. The compds. of the present invention are particularly useful in the treatment and prophylaxis of a range of conditions including cancers, protein kinase c(PKC)- or NFκB-related- or -associated conditions, cardiovascular conditions, pain, inflammatory conditions, vascular or immunol. conditions such as diabetes, neurol. conditions and infection by a range of viruses or

prokaryotic or eukaryotic organisms. The present invention further provides pharmaceutical compns. and methods of medical treatment.
REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L35 ANSWER 3 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:698214 CAPLUS
DOCUMENT NUMBER: 143:171341
TITLE: Methods for detecting infections, toxic chemicals and dietary peptides binding to lymphocyte receptors and tissue enzymes as instigators of autoimmunity in autism
INVENTOR(S): Vojdani, Aristo
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 89 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| US 2005170333 | A1 | 20050804 | US 2004-770712 | 20040203 |
| PRIORITY APPLN. INFO.: | | | US 2004-770712 | 20040203 |

AB The present invention provides methods for diagnosis and following up a prognosis of children with autism before and after treatment with different modalities administered by their clinicians, confirming the involvement of infectious agents, dietary proteins, and toxic chems. in development of autism. In particular, methods for detecting infections, toxic chems. and dietary peptides binding to lymphocyte receptors and tissue enzymes as instigators of autoimmunity in autism are described. The method utilizes detection of increased amts. of antibodies against an antigen based on infectious agent, toxic chems., or dietary proteins. Another method utilizes detection of antibodies to a self-tissue or peptide.

L35 ANSWER 4 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:1117907 CAPLUS
DOCUMENT NUMBER: 143:379850
TITLE: Diagnosis and treatment system for reward deficiency syndrome (RDS) and related behaviors
INVENTOR(S): Blum, Kenneth
PATENT ASSIGNEE(S): USA
SOURCE: U.S., 62 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| US 6955873 | B1 | 20051018 | US 2000-632838 | 20000804 |
| US 2006079495 | A1 | 20060413 | US 2005-250355 | 20051014 |
| PRIORITY APPLN. INFO.: | | | US 2000-632838 | A3 20000804 |

AB The invention concerns a kit and an intervenously administrable preparation for treatment of reward deficiency syndrome and related behavioral syndromes. An important aspect of the present invention is a kit comprising a buccal swab for obtaining a subject's DNA sample suitable for anal. of alleles associated with signal-transmitter production, reception or catabolism; and at least one composition comprising at least one of: a signal-transmitter precursor, an enhancer of precursor uptake, and an inhibitor of neurotransmitter reuptake or signal-transmitter catabolism; wherein

allelic anal. predicts a likelihood of pos. effects of a subjects intake of one or more components of the composition in effective amts. In an important aspect, the enhancer is a chromium salt, for example chromium nicotinate or chromium picolinate. Such chromium enhances certain neurotransmitter precursor uptake.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L35 ANSWER 5 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2005:1020459 CAPLUS

DOCUMENT NUMBER: 143:279463

TITLE: Metalloectopeptidase-inhibiting peptides, derived from human BPLP protein, polynucleotides and antibodies for the same, and diagnostic and therapeutic uses

INVENTOR(S): Rougeot, Catherine; Huaulme, Jean-Fancois; Ungeheuer, Marie-Noelle; Wisner, Anne; Dufour, Evelyne

PATENT ASSIGNEE(S): Institut Pasteur, Fr.

SOURCE: Eur. Pat. Appl., 38 pp.
CODEN: EPXXDW

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| EP 1577320 | A1 | 20050921 | EP 2004-290754 | 20040319 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK | | | | |
| AU 2005223492 | A1 | 20050929 | AU 2005-223492 | 20050318 |
| CA 2559215 | A1 | 20050929 | CA 2005-2559215 | 20050318 |
| WO 2005090386 | A1 | 20050929 | WO 2005-IB700 | 20050318 |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW | | | | |
| RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| EP 1725579 | A1 | 20061129 | EP 2005-708768 | 20050318 |
| R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR | | | | |

PRIORITY APPLN. INFO.: EP 2004-290754 A 20040319
WO 2005-IB700 W 20050318

OTHER SOURCE(S): MARPAT 143:279463

AB The present invention relates to peptides derived from human Basic Proline-rich Lacrimal Protein (BPLP) protein, as new inhibitors of metalloectopeptidases. Particularly, the inventors identified a new BPLP-derived peptide, of sequence QRFSR, that is considered as the functional and structural human homolog of the SMR1 (submandibular rat protein 1)-derived pentapeptide sialorphin (QHNPR). The gene BPLP is expressed in human lacrimal and submandibular glands. The BPLP-derived peptide is a maturation product of the BPLP, and exhibits an inhibitory property against a metalloectopeptidase, especially neprilysin (NEP) and/or aminopeptidase N (APN). The QRFSR peptide was synthesized and analyzed for its capacity to inhibit the degradation of the physiol. NEP substrate, i.e. substance P. The present invention also relates to polynucleotides coding for BPLP-derived peptide, to active peptide derivs., and to antibodies directed against said peptides.

Furthermore, the present invention relates to diagnostic and therapeutic uses of human BPLP protein and inhibitory peptides derived therefrom, polynucleotides coding for human BPLP protein or peptides derived therefrom as well as antibodies directed against BPLP protein or peptides derived therefrom.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L35 ANSWER 6 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:612496 CAPLUS
DOCUMENT NUMBER: 141:152222
TITLE: Novel human G-protein coupled receptor, HGPRBMY9, expressed highly in brain and testes
INVENTOR(S): Feder, John N.; Mintier, Gabriel; Ramanathan, Chandra S.; Hawken, Donald R.; Cacace, Angela M.; Bennett, Kelly L.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 69 pp., Cont.-in-part of U.S. Ser. No. 964,923.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | | DATE |
|---|---|---|---|---|---|
| US 2004147732 | A1 | 20040729 | US 2003-680402 | | 20031007 |
| US 2003096300 | A1 | 20030522 | US 2001-964923 | | 20010926 |
| PRIORITY APPLN. INFO.: | | | US 2000-235709P | P | 20000927 |
| | | | US 2001-261775P | P | 20010116 |
| | | | US 2001-309625P | P | 20010802 |
| | | | US 2001-964923 | A2 | 20010926 |

AB The present invention describes a newly discovered human G-protein coupled receptor and its encoding polynucleotide. Also described are expression vectors, host cells, agonists, antagonists, antisense mols., and antibodies associated with the polynucleotide and/or polypeptide of the present invention. In addition, methods for treating, diagnosing, preventing, and screening for disorders associated with aberrant cell growth, neurol. conditions, urol. conditions, and diseases or disorders related to the brain and testes are illustrated. Addnl. methods for treating, diagnosing, preventing, and screening for disorders associated with Alzheimer's disease, proliferative lung disorders, and disorders associated with aberrant NFkB and/or E-selectin expression and/or function are illustrated.

L35 ANSWER 7 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:372707 CAPLUS
DOCUMENT NUMBER: 140:387046
TITLE: Protein and cDNA sequences of human G-protein coupled receptor BMSOTR, and splice variant and their uses in diagnosis and therapy
INVENTOR(S): Ramanathan, Chandra S.; Gopal, Shuba; Mintier, Gabriel A.; Feder, John
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 97 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|

US 2004086881 A1 20040506 US 2003-334360 20030312
PRIORITY APPLN. INFO.: US 2002-345706P P 20020104
US 2002-355559P P 20020206

AB The present invention describes the protein and cDNA sequences of human G-protein coupled receptor (GPCR) BMSOTR and its splice variant. Also described are expression vectors, host cells for production of BMSOTR and antibodies against BMSOTR. In addition, methods for treating, diagnosing, preventing, and screening for disorders or diseases associated with abnormal biol. activity of BMSOTR are described. Transcripts corresponding to BMSOTR were expressed at high levels in the pancreas and brain, significantly in the testis and to a lesser extent in other tissues.

L35 ANSWER 8 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1998:719249 CAPLUS
DOCUMENT NUMBER: 129:340512
TITLE: Allelic polygene diagnosis of reward deficiency syndrome and treatment
INVENTOR(S): Blum, Kenneth; Comings, David E.; Ivy, John L.
PATENT ASSIGNEE(S): Kenneth Blum, Inc., USA; Board of Regents, the University of Texas System; City of Hope National Medical Center
SOURCE: PCT Int. Appl., 663 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 9848785 | A2 | 19981105 | WO 1998-US8684 | 19980429 |
| WO 9848785 | A3 | 19990401 | | |
| W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW | | | | |
| RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG | | | | |
| CA 2288990 | A1 | 19981105 | CA 1998-2288990 | 19980429 |
| AU 9872677 | A | 19981124 | AU 1998-72677 | 19980429 |
| EP 979092 | A2 | 20000216 | EP 1998-920019 | 19980429 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI | | | | |
| JP 2002511850 | T | 20020416 | JP 1998-547364 | 19980429 |
| NO 9905257 | A | 19991227 | NO 1999-5257 | 19991028 |

PRIORITY APPLN. INFO.: US 1997-44394P P 19970429
WO 1998-US8684 W 19980429

AB A multiple additive assocns. technique was applied to 29 previously known genes whose allelic polymorphisms are markers of polygenic traits, including markers for such polygenic traits as attention deficit-hyperactivity disorder, oppositional defiant disorder, conduct disorder, learning disorders, alc. and drug addiction, hypercholesterolemia, and LDL. Correlations are provided between the predisposition to reward deficiency syndrome and alleles of a number of genes including but not limited to the dopaminergic genes, dopamine transporter gene, serotonin genes, tryptophan 2,3-hydroxylase, norepinephrine genes, catecholamine metabolizing genes, GAGA genes, cannabinoid receptor gene, nicotinic cholinergic gene, NMDA receptor gene, enkephalin genes, androgen receptor gene, interferon γ gene, serotonin receptor genes, catechol O-methyltransferase gene, neuronal nitric oxide synthase gene, apolipoprotein D gene, and

uncoupling protein genes. Specific allele polymorphisms are provided as indicators of behavioral disorders as well as the likelihood of successful treatment. Enhancement of attentional processing is attained by administration of an endorphinase inhibitor or enkephalinase inhibitor and optionally, a dopamine precursor, or a serotonin precursor, a GABA precursor, or an endorphin or enkephalinase releaser, or certain herbal compds. including Rhodiola rosea extract (Pharmaline) and/or Huperzine. These components promote restoration of normal neurotransmitter function and the components combined enhance the release of dopamine at the nucleus accumbens and are non-addictive. Use of the dopamine precursors L-phenylalanine, or L-tyrosine, the enkephalinase inhibitor D-phenylalanine, and/or the serotonin precursor 5-hydroxytryptophan and a natural acetylcholenesterase inhibitor and chromium salts (i.e. picolinate, nicotinate, etc.) is especially preferred, but not limited to assist in relieving symptoms associated with brain phenylalanine deficiency.

L35 ANSWER 9 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1997:229712 CAPLUS
DOCUMENT NUMBER: 126:262727
TITLE: Hyperserotoninemia and serotonin receptor antibodies in children with autism but not mental retardation
AUTHOR(S): Singh, Vijendra K.; Singh, Edith A.; Warren, Reed P.
CORPORATE SOURCE: Immunology Laboratory, Center for Persons with Disabilities, Utah State University, Logan, UT, USA
SOURCE: Biological Psychiatry (1997), 41(6), 753-755
CODEN: BIPCBF; ISSN: 0006-3223
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Serotonin levels and brain serotonin receptor antibodies in were measured in plasmas of autistic, mentally retarded, and normal children. The authors found that autistic children had significantly higher serotonin levels and serotonin receptor antibodies.

L35 ANSWER 10 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1996:16849 CAPLUS
DOCUMENT NUMBER: 124:135428
TITLE: Low-dose naltrexone effects on plasma chemistries and clinical symptoms in autism: A double-blind, placebo-controlled study
AUTHOR(S): Bouvard, Manuel P.; Leboyer, Marion; Launay, Jean-Marie; Recasens, Christophe; Plumet, Marie-Helene; Waller-Perotte, Delphine; Tabuteau, Francois; Bondoux, Dominique; Dugas, Michel; et al.
CORPORATE SOURCE: Service de Psychopathologie de l'Enfant et de l'Adolescent, Hopital Robert Debre, Paris, 75019, Fr.
SOURCE: Psychiatry Research (1995), 58(3), 191-201
CODEN: PSRSDR; ISSN: 0165-1781
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The effect of month-long naltrexone (NTX) treatment at a daily oral dose of 0.5 mg/kg/day was contrasted with placebo (PLC) in a double-blind study with conjoint clin. and biochem. evaluations of therapeutic effects. Modest clin. benefits were achieved with both PLC and NTX, with marginally better overall results following NTX, and degree of improvement appeared to be related to plasma chemical profiles. Massively elevated levels of β-endorphin were observed in all children with assays using C-terminal antibody but not with an N-terminal antibody assay. In addition, 70% of the children exhibited abnormally low levels of adrenocorticotropic hormone, and smaller subsets exhibited elevated

norepinephrine (60%), arginine-vasopressin (50%), and serotonin (20%). The best clin. responders exhibited the clearest normalization of the elevated plasma chemistries, especially in C-terminal-β-endorphin and serotonin. There was some evidence of therapeutic carry-over effects in both clin. and biochem. measures in those children who received NTX before PLC. The results suggest that NTX only benefits a subgroup of autistic children, who may be identified by the presence of certain plasma abnormalities. These results suggest a possible linkage between abnormal plasma chemistries, especially those related to the pro-opiomelanocortin system, and autistic symptoms.

L35 ANSWER 11 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1991:551287 CAPLUS
DOCUMENT NUMBER: 115:151287
TITLE: Serotonin binding sites. II. Muramyl dipeptide binds to serotonin binding sites on myelin basic protein, LH-RH, and MSH-ACTH 4-10
AUTHOR(S): Root-Bernstein, Robert Scott; Westall, Fred C.
CORPORATE SOURCE: Dep. Physiol., Michigan State Univ., East Lansing, MI, 48824, USA
SOURCE: Brain Research Bulletin (1990), 25(6), 827-41
CODEN: BRBUDU; ISSN: 0361-9230
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The existence of structurally similar serotonin binding sites on myelin basic protein, HRH, and MSH-ACTH 4-10 has been reported. This report shows that the adjuvant peptide, muramyl dipeptide also binds to these sites. This observation may help to explain previous observations of serotonin-like activity by muramyl peptides, including the promotion of slow-wave sleep and fever induction. The observation may also provide an important link between the immune system and the nervous system that may explain the role of muramyl dipeptide adjuvants in causing autoimmune diseases to serotinin-regulated proteins and their receptors, as well as the alterations in serotonin levels that are often observed in autoimmune diseases. The observation provides concrete evidence for a dual-antigen hypothesis for the induction of autoimmune diseases by an adjuvant-peptide complex. Application of such a mechanism for induction of autoimmunity may be of importance in understanding a number of postinfectious and postvaccinal neuropathies, and suggests a possible etiol. for autism, in which many patients have high blood serotonin levels, autoimmune reactions to myelin basic protein, and antibodies to serotonin binding sites. Finally, the observation suggests that glycopeptides may act as neurotransmitters.

L35 ANSWER 12 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1985:106985 CAPLUS
DOCUMENT NUMBER: 102:106985
TITLE: Demonstration of inter- and intraspecies differences in serotonin binding sites by antibodies from an autistic child
AUTHOR(S): Todd, Richard D.; Ciaranello, Roland D.
CORPORATE SOURCE: Sch. Med., Stanford Univ., Stanford, CA, 94305, USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (1985), 82(2), 612-16
CODEN: PNASA6; ISSN: 0027-8424
DOCUMENT TYPE: Journal
LANGUAGE: English
AB 5-HT [50-67-9]-binding sites from human cerebral cortex possess pharmacol. properties that follow the same subclassification scheme as for 5-HT receptors of bovine and rat cortex. In addition, solubilized 5-HT1 and 5-HT3 sites from all 3 species have a sedimentation coefficient value of 3.4. Despite these similar pharmacol. and phys. characteristics, there are antigenic differences between receptor types and species. Human 5-HT1A sites can be distinguished from human 5-HT1B, 5-HT2, and 5-HT3 sites and

from equivalent sites in rat and bovine cortex. The anti-human 5-HT1A antibodies were discovered in the blood of an autistic child and may have clin. or etiol. significance for this disorder.

=> (autism or autistic) and (antigen or antibody) and (dynorphin or dipeptidylpeptidase IV or enkephelin)
L36 3 FILE CAPLUS
L37 0 FILE BIOTECHNO
L38 0 FILE COMPENDEX
L39 0 FILE ANABSTR
L40 0 FILE CERAB
L41 0 FILE METADEX
L42 23 FILE USPATFULL

TOTAL FOR ALL FILES
L43 26 (AUTISM OR AUTISTIC) AND (ANTIGEN OR ANTIBODY) AND (DYNORPHIN OR DIPEPTIDYLPEPTIDASE IV OR ENKEPHELIN)

=> dup rem
ENTER L# LIST OR (END):l36
PROCESSING COMPLETED FOR L36
L44 3 DUP REM L36 (0 DUPLICATES REMOVED)

=> d l44 ibib abs total

L44 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:698214 CAPLUS
DOCUMENT NUMBER: 143:171341
TITLE: Methods for detecting infections, toxic chemicals and dietary peptides binding to lymphocyte receptors and tissue enzymes as instigators of autoimmunity in autism
INVENTOR(S): Vojdani, Aristo
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 89 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| US 2005170333 | A1 | 20050804 | US 2004-770712 | 20040203 |
| PRIORITY APPLN. INFO.: | | | US 2004-770712 | 20040203 |

AB The present invention provides methods for diagnosis and following up a prognosis of children with autism before and after treatment with different modalities administered by their clinicians, confirming the involvement of infectious agents, dietary proteins, and toxic chems. in development of autism. In particular, methods for detecting infections, toxic chems. and dietary peptides binding to lymphocyte receptors and tissue enzymes as instigators of autoimmunity in autism are described. The method utilizes detection of increased amts. of antibodies against an antigen based on infectious agent, toxic chems., or dietary proteins. Another method utilizes detection of antibodies to a self-tissue or peptide.

L44 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:842682 CAPLUS
DOCUMENT NUMBER: 143:404331
TITLE: Heat shock protein and gliadin peptide promote development of peptidase antibodies in children with autism and patients with

autoimmune disease. [Erratum to document cited in CA141:205490]

AUTHOR(S): Vojdani, Aristo; Bazargan, Mohsen; Vojdani, Elroy; Samadi, John; Nourian, Alen A.; Eghbalieh, Navid; Cooper, Edwin L.

CORPORATE SOURCE: Laboratory of Comparative Neuroimmunology, Department of Neurobiology, David Geffen School of Medicine at UCLA, University of California, Los Angeles, CA, 90095, USA

SOURCE: Clinical and Diagnostic Laboratory Immunology (2005), 12(8), 1011
CODEN: CDIMEN; ISSN: 1071-412X

PUBLISHER: American Society for Microbiology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The correct article byline is given. The work was performed entirely at Immunosciences Laboratory, Inc., not at UCLA.

L44 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:496904 CAPLUS

DOCUMENT NUMBER: 141:205490

TITLE: Heat shock protein and gliadin peptide promote development of peptidase antibodies in children with autism and patients with autoimmune disease

AUTHOR(S): Vojdani, Aristo; Bazargan, Mohsen; Vojdani, Elroy; Samadi, John; Nourian, Alen A.; Eghbalieh, Navid; Cooper, Edwin L.

CORPORATE SOURCE: Laboratory of Comparative Neuroimmunology, Department of Neurobiology, David Geffen School of Medicine, University of California, Los Angeles, CA, 90095, USA

SOURCE: Clinical and Diagnostic Laboratory Immunology (2004), 11(3), 515-524
CODEN: CDIMEN; ISSN: 1071-412X

PUBLISHER: American Society for Microbiology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Searching for a mechanism underlying autoimmunity in autism, the authors postulated that gliadin peptides, heat shock protein 60 (HSP-60), and streptokinase (SK) bind to different peptidases resulting in autoantibody production against these components. The authors assessed this hypothesis in patients with autism and in those with mixed connective tissue diseases. Associated with anti-gliadin and anti-HSP antibodies, children with autism and patients with autoimmune disease developed anti-dipeptidylpeptidase I (DPP I), anti-dipeptidylpeptidase IV (DPP IV [or CD26]), and anti-aminopeptidase N (CD13) autoantibodies. A percentage of autoimmune and autistic sera were associated with elevated IgG, IgM, or IgA antibodies against 3 peptidases, gliadin, and HSP-60. These antibodies are specific, since immune absorption demonstrated that only specific antigens (e.g., DPP IV absorption of anti-DPP IV), reduced IgG, IgM, and IgA antibody levels. For direct demonstration of SK, HSP-60, and gliadin peptide binding to DPP IV, microtiter wells coated with DPP IV were reacted with SK, HSP-60, and gliadin. They were then reacted with anti-DPP IV or anti-SK, anti-HSP, and antigliadin antibodies. Adding SK, HSP-60, and gliadin peptides to DPP IV resulted in 27-43% inhibition of the DPP IV-anti-DPP IV reaction, but DPP IV-pos. peptides caused 18-20% enhancement of antigen-antibody reactions. The authors propose that (1) superantigens (e.g., SK and HSP-60) and dietary proteins (e.g., gliadin peptides) in individuals with predisposing HLA mols. bind to aminopeptidases and (2) they induce autoantibodies to peptides and tissue antigens. Dysfunctional membrane peptidases and autoantibody production may result in neuroimmune dysregulation and autoimmunity.

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REFERENCE COUNT:          64     THERE ARE 64 CITED REFERENCES AVAILABLE FOR THIS
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=> (autism or autistic) and (antigen or antibody) and (dynorphin or
dipeptidylpeptidase IV or enkephelin)
L45             0 FILE AGRICOLA
L46             0 FILE BIOTECHNO
L47             0 FILE CONFSCI
L48             0 FILE HEALSAFE
L49             0 FILE IMSDRUGCONF
L50             1 FILE LIFESCI
L51             0 FILE PASCAL

TOTAL FOR ALL FILES
L52             1 (AUTISM OR AUTISTIC) AND (ANTIGEN OR ANTIBODY) AND (DYNORPHIN
                  OR DIPEPTIDYLPEPTIDASE IV OR ENKEPHELIN)
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=> d l52 ibib abs total

L52 ANSWER 1 OF 1 LIFESCI COPYRIGHT 2007 CSA on STN
ACCESSION NUMBER: 2005:1197 LIFESCI
TITLE: Heat Shock Protein and Gliadin Peptide Promote Development of Peptidase Antibodies in Children with Autism and Patients with Autoimmune Disease
AUTHOR: Vojdani, A.*; Bazargan, M.; Vojdani, E.; Samadi, J.; Nourian, A.A.; Eghbalieh, N.; Cooper, E.L.
CORPORATE SOURCE: Section of Neuroimmunology, Immunosciences Lab., Inc., 8693 Wilshire Blvd., Suite 200, Beverly Hills, CA 90211; E-mail: immunsci@ix.netcom.com
SOURCE: Clinical and Diagnostic Laboratory Immunology [Clin. Diagn. Lab. Immunol.], (20040500) vol. 11, no. 3, pp. 515-524. ISSN: 1071-412X.
DOCUMENT TYPE: Journal
FILE SEGMENT: F
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Searching for a mechanism underlying autoimmunity in autism, we postulated that gliadin peptides, heat shock protein 60 (HSP-60), and streptokinase (SK) bind to different peptidases resulting in autoantibody production against these components. We assessed this hypothesis in patients with autism and in those with mixed connective tissue diseases. Associated with antigliadin and anti-HSP antibodies, children with autism and patients with autoimmune disease developed anti-dipeptidylpeptidase I (DPP I), anti-dipeptidylpeptidase IV (DPP IV (or CD26)) and anti-aminopeptidase N (CD13) autoantibodies. A significant percentage of autoimmune and autistic sera were associated with elevated immunoglobulin G (IgG), IgM, or IgA antibodies against three peptidases, gliadin, and HSP-60. These antibodies are specific, since immune absorption demonstrated that only specific antigens (e.g., DPP IV absorption of anti-DPP IV), significantly reduced IgG, IgM, and IgA antibody levels. For direct demonstration of SK, HSP-60, and gliadin peptide binding to DPP IV, microtiter wells coated with DPP IV were reacted with SK, HSP-60, and gliadin. They were then reacted with anti-DPP IV or anti-SK, anti-HSP, and antigliadin antibodies . Adding SK, HSP-60, and gliadin peptides to DPP IV resulted in 27 to 43% inhibition of the DPP IV-anti- DPP IV reaction, but DPP IV-positive peptides caused 18 to 20% enhancement of antigen-antibody reactions. We propose that (i) superantigens (e.g., SK and HSP- 60) and dietary proteins (e.g., gliadin peptides) in individuals with predisposing HLA molecules bind to aminopeptidases and (ii) they induce autoantibodies to peptides and tissue antigens. Dysfunctional membrane peptidases and autoantibody production may result in neuroimmune dysregulation and autoimmunity.

=> (autism or autistic) and (antigen or antibody) and (enkephelin or substance P or somatostatin or serotonin or serotonin receptor)
L53 0 FILE AGRICOLA
L54 1 FILE BIOTECHNO
L55 0 FILE CONFSCI
L56 0 FILE HEALSAFE
L57 0 FILE IMSDRUGCONF
L58 2 FILE LIFESCI
L59 5 FILE PASCAL

TOTAL FOR ALL FILES
L60 8 (AUTISM OR AUTISTIC) AND (ANTIGEN OR ANTIBODY) AND (ENKEPHELIN OR SUBSTANCE P OR SOMATOSTATIN OR SEROTONIN OR SEROTONIN RECEPTOR)

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=> dup rem
ENTER L# LIST OR (END):l60
DUPLICATE IS NOT AVAILABLE IN 'IMSDRUGCONF'.
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L61            6 DUP REM L60 (2 DUPLICATES REMOVED)

=> d l61 ibib abs total

L61  ANSWER 1 OF 6  LIFESCI    COPYRIGHT 2007 CSA on STN
ACCESSION NUMBER:   2006:94702  LIFESCI
TITLE:              A Family Based Linkage Analysis of HLA and 5-HTTLPR Gene
                    Polymorphisms in Sardinian Children with Autism
                    Spectrum Disorder
AUTHOR:             Guerini, Franca R.; Manca, Salvatorica; Sotgiu, Stefano;
                    Tremolada, Sara; Zanzottera, Milena; Agliardi, Cristina;
                    Zanetta, Lorenzo; Saresella, Marina; Mancuso, Roberta; De
                    Silvestri, Annalisa; Fois, Maria Laura; Arru, Giannina;
                    Ferrante, Pasquale
CORPORATE SOURCE:   Laboratory of Molecular Medicine and Biotechnologies, Don
                    C. Gnocchi Foundation IRCCS, S. Maria Nascente, Milan,
                    Italy; E-mail: pferrante@dongnocchi.it
SOURCE:             Human Immunology [Hum. Immunol.], (20060200) vol. 67, no.
                    1-2, pp. 108-117.
                    ISSN: 0198-8859.
DOCUMENT TYPE:      Journal
FILE SEGMENT:       F
LANGUAGE:           English
SUMMARY LANGUAGE:   English
AB   Autism spectrum disorders (ASD) are characterized by a broad
     range in clinical presentation. Although a definite genetic cause has not
     yet been fully demonstrated, family based studies suggest that a
     multigenic pattern may be responsible for susceptibility, but most results
     are conflicting and have yet to be replicated. The purpose of this
     investigation was to analyze the linkage of the human leukocyte
     antigen (HLA) and the human serotonin transporter coding
     (5- HTTLPR) genes with ASD in a group of 37 families of Sardinian
     ethnicity in insular Italy. In 50% of these families, ASD is linked to
     HLA, and in the other 50% it is linked to 5-HTTLPR polymorphic genes; in
     other words, linkage to one or the other was evident in all cases. Despite
     a very homogenous genetic pattern being generally reported for Sardinians,
     the linkage observed with HLA and 5- HTTLPR genetic regions indicated a
     statistically defined heterogeneity (p = 0.002). No allelic HLA or
     5-HTTLPR polymorphisms were specifically associated with ASD, suggesting
     these loci as markers of other genes mapped in their close proximity that
     may be more directly involved and thus may merit further analytical
     studies.

L61   ANSWER 2 OF 6  PASCAL  COPYRIGHT 2007 INIST-CNRS. ALL RIGHTS RESERVED. on
      STN
ACCESSION NUMBER:         2004-0073160   PASCAL
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                          reserved.
TITLE (IN ENGLISH):       A hypothalamic digoxin-mediated model for
                          autism
AUTHOR:                   RAVI KUMAR KURUP; PARAMESWARA ACHUTHA KURUP
CORPORATE SOURCE:         Department of Neurology, Medical College Hospital,
                          Trivandrum, Kerala, India; Metabolic Disorders
                          Research Center, Trivandrum, Kerala, India
SOURCE:                   International journal of neuroscience, (2003),
                          113(11), 1537-1559, refs. 2 p.1/4
                          ISSN: 0020-7454  CODEN: IJNUB7
DOCUMENT TYPE:            Journal
BIBLIOGRAPHIC LEVEL:      Analytic
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COUNTRY: United Kingdom
LANGUAGE: English
AVAILABILITY: INIST-4537, 354000114835190060
AN 2004-0073160 PASCAL

AB The isoprenoid pathway and its metabolites-digoxin, dolichol, and ubiquinone-were assessed in autism. The isoprenoid pathway and digoxin status was also studied for comparison in individuals of differing hemispheric dominance to determine the role of cerebral dominance in the genesis of autism. There was an upregulation of the isoprenoid pathway as evidenced by elevated HMG CoA reductase activity in autism. Digoxin, an endogenous Na.sup.+-K.sup.+ ATPase inhibitor secreted by the hypothalamus, was found to be elevated and RBC membrane Na.sup.+-K.sup.+ ATPase activity was found to be reduced in autism. Membrane Na.sup.+-K.sup.+ ATPase inhibition can result in increased intracellular Ca.sup.2.sup.+ and reduced magnesium levels. Hypothalamic digoxin can modulate conscious and subliminal perception and its dysfunction may lead to autism. Digoxin can also preferentially upregulate tryptophan transport over tyrosine resulting in increased levels of depolarizing tryptophan catabolite-serotonin, quinolinic acid (NMDA agonist), strychnine (blocks glycinergic inhibitory transmission), and nicotine (promotes dopamine release) and decreased levels of hyperpolarizing tyrosine catabolites-dopamine, noradrenaline, and morphine-contributing to membrane Na.sup.+-K.sup.+ ATPase inhibition. Increased nicotine levels can produce increased dopaminergic transmission in the presence of low dopamine levels. NMDA excitotoxicity could result from hypomagnesemia induced by membrane Na.sup.+-K.sup.+ ATPase inhibition and quinolinic acid, an NMDA agonist acting on the NMDA receptor. Hypomagnesemia and increased dolichol level can affect glycoconjugate metabolism and membranogenesis leading on to disordered synaptic connectivity in the limbic allocortex and defective presentation of viral antigens and neuronal antigens contributing to autoimmunity and viral persistance important in the pathogenesis. Membrane Na.sup.+-K.sup.+ ATPase inhibition can produce immune activation, a component of autoimmunity. Mitochondrial dysfunction consequent to altered calcium/magnesium ratios and reduced ubiquinone levels can result in increased free radical generation and reduced free radical scavenging and defective apoptosis leading to abnormal synaptogenesis. Autism can thus be considered a syndrome of hypothalamic digoxin hypersecretion consequent to an upregulated isoprenoid pathway. The biochemical patterns including hyperdigoxinemia observed in autism correlated with those obtained in right hemispheric chemical dominance. Right hemispheric chemical dominance is a predisposing factor for autism.

L61 ANSWER 3 OF 6 PASCAL COPYRIGHT 2007 INIST-CNRS. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 1997-0235940 PASCAL

TITLE (IN ENGLISH): Hyperserotoninemia and serotonin receptor antibodies in children with autism but not mental retardation
AUTHOR: SINGH V. K.; SINGH E. A.; WARREN R. P.
CORPORATE SOURCE: Immunology Laboratory, Center for Persons with Disabilities, Utah State University, Logan, Utah, United States
SOURCE: Biological psychiatry : (1969), (1997), 41(6), 753-755, 11 refs.
ISSN: 0006-3223 CODEN: BIPCBF
DOCUMENT TYPE: Journal; Short communication
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English

AVAILABILITY: INIST-11378, 354000063585020180
AN 1997-0235940 PASCAL
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L61 ANSWER 4 OF 6 PASCAL COPYRIGHT 2007 INIST-CNRS. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 1995-0592064 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 1995 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Low-dose naltrexone effects on plasma chemistries and clinical symptoms in autism : a double-blind, placebo-controlled study
AUTHOR: BOUVARD M. P.; LEBOYER M.; LAUNAY J.-M.; RECASENS C.; PLUMET M.-H.; WALLER-PEROTTE D.; TABUTEAU F.; BONDOUX D.; DUGAS M.; LENSING P.; PANKSEPP J.
CORPORATE SOURCE: Hop. Robert Debre, serv. psychopathologie enfant adolescent, 75019 Paris, France; Hop. Pitie Salpetriere, serv. psychiatrie adulte, 75013 Paris, France; Hop. Saint Louis, lab. neurochimie communications cellulaires, 75010 Paris, France
SOURCE: Psychiatry research, (1995), 58(3), 191-201, refs. 1 p.1/4
ISSN: 0165-1781 CODEN: PSRSDR
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: Ireland
LANGUAGE: English
AVAILABILITY: INIST-18303, 354000050442690020
AN 1995-0592064 PASCAL
CP Copyright .COPYRGT. 1995 INIST-CNRS. All rights reserved.
AB The effect of month-long naltrexone (NTX) treatment at a daily oral dose of 0.5 mg/kg/day was contrasted with placebo (PLC) in a double-blind study with conjoint clinical and biochemical evaluations of therapeutic effects. Modest clinical benefits were achieved with both PLC and NTX, with marginally better overall results following NTX, and degree of improvement appeared to be related to plasma chemical profiles. Massively elevated levels of β-endorphin were observed in all children with assays using C-terminal antibody but not with an N-terminal antibody assay. In addition, 70% of the children exhibited abnormally low levels of adrenocorticotropic hormone, and smaller subsets exhibited elevated norepinephrine (60%), arginine-vasopressin (50%), and serotonin (20%). The best clinical responders exhibited the clearest normalization of the elevated plasma chemistries, especially in C-terminal-β-endorphin and serotonin. There was some evidence of therapeutic carry-over effects in both clinical and biochemical measures in those children who received NTX before PLC. The results suggest that NTX only benefits a subgroup of autistic children, who may be identified by the presence of certain plasma abnormalities. These results suggest a possible linkage between abnormal plasma chemistries, especially those related to the pro-opiomelanocortin system, and autistic symptoms.

L61 ANSWER 5 OF 6 PASCAL COPYRIGHT 2007 INIST-CNRS. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 1992-0310842 PASCAL
TITLE (IN ENGLISH): Hyperserotoninemia and antiserotonin antibodies in autism and other disorders
AUTHOR: YUWILER A.; JEAN CHEN SHIH; CHONG-HONG CHEN; RITVO E. R.; HANNA G.; ELLISON G. W.; KING B. H.
CORPORATE SOURCE: Univ. California-Los Angeles-school medicine, veterans administration West Los Angeles medical cent., United States
SOURCE: Journal of autism and developmental disorders, (1992),

22(1), 33-45, refs. 1 p.
ISSN: 0162-3257 CODEN: JADDDQ
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-15018, 354000021114830030
AN 1992-0310842 PASCAL
AB This study examined the linkage between elevated blood serotonin in autism and the presence of circulating autoantibodies against the serotonin 5HT.sub.1.sub.A receptor. Information was also obtained on the diagnostic and receptor specificity of these autoantibodies. Blood serotonin was measured as was inhibition of serotonin binding to human cortical membranes by antibody-rich fractions of blood from controls and from patients with childhood autism, schizophrenia, obsessive-compulsive disorder, Tourette's and multiple sclerosis

L61 ANSWER 6 OF 6 BIOTECHNO COPYRIGHT 2007 Elsevier Science B.V. on STN
DUPLICATE
ACCESSION NUMBER: 1985:15128319 BIOTECHNO
TITLE: Demonstration of inter- and intraspecies differences in serotonin binding sites by antibodies from an autistic child
AUTHOR: Todd R.D.; Ciaranello R.D.
CORPORATE SOURCE: Laboratory of Developmental Neurochemistry, Division of Child Psychiatry, Department of Psychiatry and Behavioral Sciences, Stanford University School of Medicine, Stanford, CA 94305, United States.
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1985), 82/2 (612-616)
CODEN: PNASA6
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
AN 1985:15128319 BIOTECHNO
AB Serotonin (5-HT) binding sites from bovine and rat cerebral cortex membranes share pharmacological properties that allow both to be subclassified by the same criteria. We show here that ¢.sup.3H!5-HT binding sites from human cortex also possess pharmacological properties that follow the same subclassification scheme as for bovine and rat cortex. In addition, we show that solubilized 5-HT.sub.1 and 5-HT.sub.3 sites from all three species have an s(20,w) value of 3.4. Despite these similar pharmacological and physical characteristics, we can demonstrate antigenic differences between receptor types and species. Human 5-HT(1A) sites can be distinguished from human 5-HT(1B), 5-HT.sub.2, and 5-HT.sub.3 sites and from equivalent sites in rat and bovine cortex. The anti-human 5-HT(1A) antibodies were discovered in the blood of an autistic child and may have clinical or etiologic significance for this disorder.

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L62 17 FILE CAPLUS
L63 9 FILE BIOSIS
L64 8 FILE MEDLINE
L65 11 FILE EMBASE
L66 403 FILE USPATFULL

TOTAL FOR ALL FILES
L67 448 (AUTISM OR AUTISTIC) AND (ANTIGEN OR ANTIBODY) AND (CD13 OR CD26 OR TRANSGLUTAMINASE OR DPPI OR DIPEPTIDYL PEPTIDASE IV OR SECRETIN OR GASTRIN OR MOTILIN)

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PROCESSING COMPLETED FOR L63
PROCESSING COMPLETED FOR L64
PROCESSING COMPLETED FOR L65
L68 30 DUP REM L62-65 (15 DUPLICATES REMOVED)

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L68 ANSWER 1 OF 30 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2006:1205478 CAPLUS
DOCUMENT NUMBER: 145:503524
TITLE: Identification of diagnostic biomarkers for autism by multiplatform analysis of blood cell immunophenotype and serum protein and metabolite content
INVENTOR(S): Amaral, David G.; Corbett, Blythe A.
PATENT ASSIGNEE(S): The Regents of the University of California, USA
SOURCE: PCT Int. Appl., 82pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2006121952 | A2 | 20061116 | WO 2006-US17581 | 20060505 |

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,

IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

US 2007003922 A1 20070104 US 2006-381976 20060505

PRIORITY APPLN. INFO.: US 2005-678865P P 20050505

AB The present invention is directed to methods for diagnosing neurodevelopmental disorders, including autism, by employing a multiplatform anal. of blood cell immunophenotype and serum polypeptide and metabolite content. The present invention provides methods of identifying biomarkers indicative of the presence of a neurodevelopmental disorder, including an autism spectrum disorder, in an individual, using cytometry and mass spectrometry. The invention further provides methods of using the identified biomarkers to diagnose the presence of a neurodevelopmental disorder, including an autism spectrum disorder.

L68 ANSWER 2 OF 30 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2006263569 EMBASE

TITLE: [Atypical celiac disease in risk group in children]. ATYPOWA CELIAKIA W GRUPACH RYZYKA U DZIECI.

AUTHOR: Burda-Muszynska B.; Oralewska B.; Cukrowska B.; Zielinska-Michalkiewicz M.; Olszaniecka M.; Stolarczyk A.; Socha J.

CORPORATE SOURCE: Dr. B. Burda-Muszynska, Klinika Gastroenterologii, Hepatologii i Immunologii, Instytut Pomnik - Centrum Zdrowia Dziecka, Aleja Dzieci Polskich 20, 04-734 Warszawa, Poland

SOURCE: Pediatria Wspolczesna, (2006) Vol. 8, No. 2, pp. 99-102. . Refs: 24
ISSN: 1507-5532 CODEN: PWESBM

COUNTRY: Poland

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 007 Pediatrics and Pediatric Surgery
048 Gastroenterology

LANGUAGE: Polish

SUMMARY LANGUAGE: Polish; English

ENTRY DATE: Entered STN: 20 Jun 2006
Last Updated on STN: 20 Jun 2006

AB Introduction: During last 20 years clinical course of celiac disease changed mostly into atypical, often with clinical manifestation outside of the gastrointestinal tract. Objectives: The aim of the study was to evaluate the prevalence of atypical celiac disease in risk groups of children. Material and methods: We studied 645 children aged 7 months-17.5 years with irritable bowel syndrome (IBS) (n=219), epilepsy (n=74), attention-deficit/hyperactivity disorder (ADHD) (n=93), autism (n=62), short stature and low weight (n=112) and the group with other symptoms (n=85) including anaemia, autoimmune hepatitis, idiopathic hypertransaminaesemia, cirrhosis and osteoporosis. In all patients immunofluorescence anti-endomysium (EmA) and immunoenzymatic anti-transglutaminase (tTG) antibody tests were performed. In case of positive serological tests a small bowel biopsy was taken for histological analyses according Marsh scale. Results: Positive screening tests were found in 11 children (the prevalence 1:58) having symptoms of IBS (n=4, 1:54), ADHD (n=3, 1:31), short stature and low weight (n=2, 1:56), anaemia (n=1), cirrhosis (n=1). Histological analysis showed signs of mucosa damaged in 8 out of 10 analyzed biopsies. Normal mucosa was found in 2 children with ADHD. Five children presented Marsh III lesions, one Marsh II and two Marsh I. Children with Marsh III were affected by IBS (n=3), cirhosis (n=1) and anaemia (n=1), with Marsh II by IBS (n=1), with Marsh I by ADHD and short stature and low weight. Conclusions: Due to the high prevalence of atypical celiac disease, screening test should be performed at least in risk groups of children.

L68 ANSWER 3 OF 30 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:962041 CAPLUS
DOCUMENT NUMBER: 143:242034
TITLE: DPP-IV inhibitors for neurodegenerative and cognitive disorders
INVENTOR(S): Hughes, Thomas Edward
PATENT ASSIGNEE(S): Novartis A.-G., Switz.; Novartis Pharma G.m.b.H.
SOURCE: PCT Int. Appl., 44 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2005079795 | A2 | 20050901 | WO 2005-EP1729 | 20050218 |
| WO 2005079795 | A3 | 20051110 | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, SM | | | | |
| RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| AU 2005215136 | A1 | 20050901 | AU 2005-215136 | 20050218 |
| CA 2555399 | A1 | 20050901 | CA 2005-2555399 | 20050218 |
| EP 1732550 | A2 | 20061220 | EP 2005-707520 | 20050218 |
| R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR | | | | |

PRIORITY APPLN. INFO.: US 2004-546229P P 20040220
US 2004-607902P P 20040908
WO 2005-EP1729 W 20050218

AB The invention relates to the use of a dipeptidyl peptidase IV inhibitor (DPP-IV inhibitor) or a pharmaceutically acceptable salt thereof for the prevention, delay of progression or the treatment of neurodegenerative disorders, cognitive disorders and for improving memory (both short term and long term) and learning ability.

L68 ANSWER 4 OF 30 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:281768 CAPLUS
DOCUMENT NUMBER: 142:354292
TITLE: Use of proline-specific endoproteases to hydrolyse proline-rich peptides at acid pH in food processing
INVENTOR(S): Edens, Luppo; Van Der Hoeven, Robertus Antonius Mijndert; De Roos, Andre Leonardus; Harvey, Melissa
PATENT ASSIGNEE(S): DSM IP Assets B.V., Neth.
SOURCE: PCT Int. Appl., 56 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2005027953 | A2 | 20050331 | WO 2004-EP10782 | 20040923 |

WO 2005027953 A3 20050616
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
EP 1663298 A2 20060607 EP 2004-765616 20040923
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK
PRIORITY APPLN. INFO.: EP 2003-78012 A 20030923
EP 2003-78496 A 20031106
WO 2004-EP10782 W 20040923

AB A method of processing protein-rich food products, such as milk, to eliminate proline-rich peptides uses prolyl oligopeptidases to hydrolyze such peptides on the C-terminal side of the proline. This is particularly useful in treating milk products, such as caseins, to eliminate casomorphins. The enzymes have an acid pH optimum and are members of the subtilisin family, but can use these proline-rich substrates that are resistant to other serine proteinases. The enzymes also accept a limited size range of peptides (4-40 amino acids) as substrates. Removal of these peptides in food processing is useful in the prevention of psychiatric and autoimmune disorders. The enzymes may be administered orally to a patient as needed because of their acid pH optima. A proline endopeptidase of Aspergillus nigers was used in combination with the com. subtilisin Alcalase to degrade caseins. Alcalase alone generated significant quantities of β-casomorphin from β-caseins. When used in combination with the proline endopeptidase, the yield of β-casomorphin was lowered by ≥2000-fold.

L68 ANSWER 5 OF 30 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2005:698214 CAPLUS
DOCUMENT NUMBER: 143:171341
TITLE: Methods for detecting infections, toxic chemicals and dietary peptides binding to lymphocyte receptors and tissue enzymes as instigators of autoimmunity in autism
INVENTOR(S): Vojdani, Aristo
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 89 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| US 2005170333 | A1 | 20050804 | US 2004-770712 | 20040203 |

PRIORITY APPLN. INFO.: US 2004-770712 20040203

AB The present invention provides methods for diagnosis and following up a prognosis of children with autism before and after treatment with different modalities administered by their clinicians, confirming the involvement of infectious agents, dietary proteins, and toxic chems. in development of autism. In particular, methods for detecting infections, toxic chems. and dietary peptides binding to lymphocyte receptors and tissue enzymes as instigators of autoimmunity in autism are described. The method utilizes detection of increased amts. of antibodies against an antigen based on

infectious agent, toxic chems., or dietary proteins. Another method utilizes detection of antibodies to a self-tissue or peptide.

L68 ANSWER 6 OF 30 MEDLINE on STN
ACCESSION NUMBER: 2005336840 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15977319
TITLE: Novel treatments for autistic spectrum disorders.
AUTHOR: Levy Susan E; Hyman Susan L
CORPORATE SOURCE: Children's Seashore House, The Children's Hospital of Philadelphia, University of Pennsylvania School of Medicine, Philadelphia, 1914, USA.. Levys@email.chop.edu
CONTRACT NUMBER: 2 U19 HD35466 (NICHD)
CCU320394-05 (CDC)
SOURCE: Mental retardation and developmental disabilities research reviews, (2005) Vol. 11, No. 2, pp. 131-42. Ref: 189
Journal code: 9517974. ISSN: 1080-4013.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200511
ENTRY DATE: Entered STN: 1 Jul 2005
Last Updated on STN: 9 Nov 2005
Entered Medline: 8 Nov 2005

AB In no area of developmental pediatric practice is there more controversy regarding the choice of treatment than related to children with autistic spectrum disorders (ASD). Complementary and alternative medical therapies (CAM) are often elected because they are perceived as treating the cause of symptoms rather than the symptoms themselves. CAM used for autism can be divided by proposed mechanism: immune modulation, gastrointestinal, supplements that affect neurotransmitter function, and nonbiologic intervention. Secretin as a therapy for autism is discussed as an example of how a clinical observation rapidly grew to a widespread treatment before well-designed studies demonstrated absence of effect. The plausibility for behavioral effect was not substantiated by clinical studies. CAM used for treatment of autism is examined in terms of rationale, evidence of efficacy, side effects, and additional commentary. Families and clinicians need access to well-designed clinical evidence to assist them in choice of therapies.


L68 ANSWER 7 OF 30 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2005002665 EMBASE
TITLE: Transient expression of secretin in serotoninergic neurons of mouse brain during development.
AUTHOR: Lossi L.; Bottarelli L.; Candusso M.E.; Leiter A.B.; Rindi G.; Merighi A.
CORPORATE SOURCE: Dr. A. Merighi, Dept. of Veterinary Morphophysiology, University of Turin, Via Leonardo da Vinci 44, 10095 Grugliasco, Torino, Italy. adalberto.merighi@unito.it
SOURCE: European Journal of Neuroscience, (2004) Vol. 20, No. 12, pp. 3259-3269. .
Refs: 44
ISSN: 0953-816X CODEN: EJONEI
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
021 Developmental Biology and Teratology
032 Psychiatry

LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 13 Jan 2005
Last Updated on STN: 13 Jan 2005

AB Existence of the gastro-intestinal peptide secretin in the CNS has been a matter of debate, and contrasting results have been reported, altogether indicating that the CNS is not a major site of production of this peptide. A thorough analysis was conducted in brain of transgenic mice in which the expression of the early region of simian virus 40 large T antigen (Tag) is under control of the rat secretin gene promoter. We studied Tag expression in the brains of E14-P90 transgenic mice as well as secretin mRNA and protein expression in transgenic and control CD1 mice at corresponding developmental stages. We show here a perfect correspondence of Tag and secretin mRNA expression in the mesencephalon of transgenic and normal mice between E14 and birth. In embryos, Tag is also expressed in the spinal cord, as well as in several areas of the peripheral nervous system. Localization of Tag in P0-P90 animals becomes restricted to a single compact cellular mass in mesencephalon at the level of the dorsal raphe, raphe magnus and lateral paragigantocellular nuclei. Neurons of these nuclei display secretin mRNA from E14 to birth, in both control CD1 and transgenic mice. Approximately half of these secretin -expressing neurons are immunoreactive for serotonin (5HT) and/or tryptophan hydroxylase. These results demonstrate that the secretin gene is transiently expressed in mouse serotoninergic mesencephalic neurons during development. In addition our data suggest a trophic role for secretin on neurons known to be involved in multiple superior functions in the normal brain, and lost in neurodegenerative disorders.

L68 ANSWER 8 OF 30 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 2004:496904 CAPLUS
DOCUMENT NUMBER: 141:205490
TITLE: Heat shock protein and gliadin peptide promote development of peptidase antibodies in children with autism and patients with autoimmune disease
AUTHOR(S): Vojdani, Aristo; Bazargan, Mohsen; Vojdani, Elroy; Samadi, John; Nourian, Alen A.; Eghbalieh, Navid; Cooper, Edwin L.
CORPORATE SOURCE: Laboratory of Comparative Neuroimmunology, Department of Neurobiology, David Geffen School of Medicine, University of California, Los Angeles, CA, 90095, USA
SOURCE: Clinical and Diagnostic Laboratory Immunology (2004), 11(3), 515-524
CODEN: CDIMEN; ISSN: 1071-412X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Searching for a mechanism underlying autoimmunity in autism, the authors postulated that gliadin peptides, heat shock protein 60 (HSP-60), and streptokinase (SK) bind to different peptidases resulting in autoantibody production against these components. The authors assessed this hypothesis in patients with autism and in those with mixed connective tissue diseases. Associated with anti-gliadin and anti-HSP antibodies, children with autism and patients with autoimmune disease developed anti-dipeptidylpeptidase I (DPP I), anti-dipeptidylpeptidase IV (DPP IV [or CD26]), and anti-aminopeptidase N (CD13) autoantibodies. A percentage of autoimmune and autistic sera were associated with elevated IgG, IgM, or IgA antibodies against 3 peptidases, gliadin, and HSP-60. These antibodies are specific, since immune absorption demonstrated that only specific antigens (e.g., DPP IV absorption of anti-DPP IV), reduced IgG, IgM, and IgA antibody

levels. For direct demonstration of SK, HSP-60, and gliadin peptide binding to DPP IV, microtiter wells coated with DPP IV were reacted with SK, HSP-60, and gliadin. They were then reacted with anti-DPP IV or anti-SK, anti-HSP, and antigliadin antibodies. Adding SK, HSP-60, and gliadin peptides to DPP IV resulted in 27-43% inhibition of the DPP IV-anti-DPP IV reaction, but DPP IV-pos. peptides caused 18-20% enhancement of antigen-antibody reactions. The authors propose that (1) superantigens (e.g., SK and HSP-60) and dietary proteins (e.g., gliadin peptides) in individuals with predisposing HLA mols. bind to aminopeptidases and (2) they induce autoantibodies to peptides and tissue antigens. Dysfunctional membrane peptidases and autoantibody production may result in neuroimmune dysregulation and autoimmunity.

REFERENCE COUNT: 64 THERE ARE 64 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L68 ANSWER 9 OF 30 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 2

ACCESSION NUMBER: 2004:185188 CAPLUS
DOCUMENT NUMBER: 141:52075
TITLE: Secretin: Hypothalamic Distribution and Hypothesized Neuroregulatory Role in Autism
AUTHOR(S): Welch, M. G.; Keune, J. D.; Welch-Horan, T. B.; Anwar, N.; Anwar, M.; Ludwig, R. J.; Ruggiero, D. A.
CORPORATE SOURCE: Laboratories of Childhood Regulatory Disorders and Behavorial Neuroanatomy, College of Physicians and Surgeons, Division of Neuroscience, NYSPI, Columbia University, New York, NY, USA
SOURCE: Cellular and Molecular Neurobiology (2004), 24(2), 219-241
CODEN: CMNEDI; ISSN: 0272-4340
PUBLISHER: Kluwer Academic/Plenum Publishers
DOCUMENT TYPE: Journal
LANGUAGE: English

AB This study aims (1) to determine whether secretin is synthesized centrally, specifically by the HPA axis and (2) to discuss, on the basis of the findings in this and previous studies, secretin's possible neuroregulatory role in autism. An immunocytochem. technique with single-cell resolution was performed in 12 age/weight-matched male rats pretreated with stereotaxic microinjection of colchicine (0.6 μg/kg) or vehicle into the lateral ventricle. Following 2-day survival, rats were anesthetized and perfused for immunocytochem. Brain segments were blocked and alternate frozen 30-μm sections incubated in rabbit antibodies against secretin, vasoactive intestinal peptide, glucagon, or pituitary-adenylate-cyclase-activating peptide. Adjacent sections were processed for Nissl stain. Preadsorption studies were performed with members of the secretin peptide family to demonstrate primary antibody specificity. Specificity of secretin immunoreactivity (ir) was verified by clear-cut preadsorption control data and relatively high concns. and distinct topog. localization of secretin ir to paraventricular/supraoptic and intercalated hypothalamic nuclei. Secretin levels were upregulated by colchicine, an exemplar of homeostatic stressors, as compared with low constitutive expression in untreated rats. This study provides the first direct immunocytochem. demonstration of secretinergic immunoreactivity in the forebrain and offers evidence that the hypothalamus, like the gut, is capable of synthesizing secretin. Secretin's dual expression by gut and brain secretin cells, as well as its overlapping central distribution with other stress-adaptation neurohormones, especially oxytocin, indicates that it is stress-sensitive. A neuroregulatory relationship between the peripheral and central stress response systems is suggested, as is a dual role for secretin in conditioning both of those stress-adaptation systems. Colchicine-induced upregulation of secretin indicates that secretin may be synthesized on demand in response to stress, a

possible mechanism of action that may underlie secretin's role in autism.

REFERENCE COUNT: 136 THERE ARE 136 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L68 ANSWER 10 OF 30 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2003:397078 CAPLUS

DOCUMENT NUMBER: 138:397218

TITLE: Multi-parameter high throughput screening assays (MPHTS) for identifying therapeutic compounds for treatment of neuropsychiatric and neurodegenerative disorders

INVENTOR(S): Altar, Anthony C.; Brockman, Jeffrey A.; Evans, David; Hook, Derek; Klimczak, Leszek; Laeng, Pascal; Palfreyman, Michael; Rajan, Prithi

PATENT ASSIGNEE(S): Psychiatric Genomics, Inc., USA

SOURCE: PCT Int. Appl., 103 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 2

PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | | DATE |
|---|---|---|---|---|---|
| WO 2003042654 | A2 | 20030522 | WO 2002-US31106 | | 20020927 |
| WO 2003042654 | A9 | 20030807 | | | |
| WO 2003042654 | A3 | 20040603 | | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW | | | | | |
| RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | | |
| US 2003096264 | A1 | 20030522 | US 2002-175523 | | 20020618 |
| AU 2002368043 | A1 | 20040123 | AU 2002-368043 | | 20020618 |
| AU 2002327791 | A1 | 20030526 | AU 2002-327791 | | 20020927 |

PRIORITY APPLN. INFO.:

| | | |
|---|---|---|
| US 2001-333047P | P | 20011114 |
| US 2002-349936P | P | 20020118 |
| US 2002-361834P | P | 20020304 |
| US 2002-175523 | A | 20020618 |
| US 2001-299151P | P | 20010618 |
| US 2001-317828P | P | 20010907 |
| US 2001-325150P | P | 20010925 |
| WO 2002-US19457 | W | 20020618 |
| WO 2002-US31106 | W | 20020927 |

AB The present invention relates to screening methods and assays that are referred to herein as multi-parameter high throughput screening (MPHTS) assays. These methods pertain to the combination of data generated from gene expression profiling coupled with methods for the systematic anal. and/or employment of such data. Such methods comprise steps of: identifying a plurality of disease signature genes and identifying a plurality of drug signature genes, followed by obtaining a score value for each of these genes that is a function of each gene's differential expression in the disease signature compared to its expression in the drug signature. Once scored, disease signature and drug signature genes having the highest score(s) may then ben selected as efficacy genes. Large nos. of candidate compds may be screened in vitro to identify ones that are particularly suitable and promising as novel therapeutic agents. These

MPHTS assays are useful for identifying candidate pharmaceutical compds. In particular, the screening methods of this invention may be used to identify compds. that have potential therapeutic benefits for the treatment of neuropsychiatric and neurodegenerative disorders, including schizophrenia, bipolar affective disorder (BAD), autism, and Alzheimer's disease to name a few.

L68 ANSWER 11 OF 30 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:396350 CAPLUS
DOCUMENT NUMBER: 138:396234
TITLE: Multi-parameter high throughput screening assays (MPHTS) for screening of therapeutic candidates for neuropsychiatric and neurodegenerative disorders
INVENTOR(S): Altar, C. Anthony; Brockman, Jeffrey A.; Evans, David; Hook, Derek; Klimczak, Leszek J.; Laeng, Pascal; Palfreyman, Michael; Rajan, Prithi
PATENT ASSIGNEE(S): Psychiatric Genomics, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 39 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| US 2003096264 | A1 | 20030522 | US 2002-175523 | 20020618 |
| WO 2004005882 | A2 | 20040115 | WO 2002-US19457 | 20020618 |
| WO 2004005882 | A3 | 20050127 | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, EC, EE, ES, FI, GB, GD, GE, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW | | | | |
| RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| AU 2002368043 | A1 | 20040123 | AU 2002-368043 | 20020618 |
| WO 2003042654 | A2 | 20030522 | WO 2002-US31106 | 20020927 |
| WO 2003042654 | A9 | 20030807 | | |
| WO 2003042654 | A3 | 20040603 | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW | | | | |
| RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| AU 2002327791 | A1 | 20030526 | AU 2002-327791 | 20020927 |
| US 2005181433 | A1 | 20050818 | US 2005-99266 | 20050404 |

| PRIORITY APPLN. INFO.: | | |
|---|---|---|
| US 2001-299151P | P | 20010618 |
| US 2001-317828P | P | 20010907 |
| US 2001-325150P | P | 20010925 |
| US 2001-333047P | P | 20011114 |
| US 2002-349936P | P | 20020118 |
| US 2002-361834P | P | 20020304 |
| US 2002-175523 | A | 20020618 |
| WO 2002-US19457 | W | 20020618 |
| WO 2002-US31106 | W | 20020927 |

AB The present invention relates to screening methods and assays that are referred to herein as multi-parameter high throughput screening (MPHTS) assays. These MPHTS assays are useful for identifying candidate pharmaceutical compds. In particular, the screening methods of this invention may be used to identify compds. that have potential therapeutic benefits for the treatment of neuropsychiatric and neurodegenerative disorders, including schizophrenia, bipolar affective disorder (BAD), autism and Alzheimer's disease to name a few.

L68 ANSWER 12 OF 30 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2003405199 EMBASE
TITLE: Autism.
AUTHOR: Volkmar F.R.; Pauls D.
CORPORATE SOURCE: Dr. F.R. Volkmar, Child Study Center, Yale University, PO Box 207900, New Haven, CT 06520, United States. fred.volkmar@yale.edu
SOURCE: Lancet, (4 Oct 2003) Vol. 362, No. 9390, pp. 1133-1141. . Refs: 164
ISSN: 0140-6736 CODEN: LANCAO
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 017 Public Health, Social Medicine and Epidemiology
022 Human Genetics
032 Psychiatry
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 23 Oct 2003
Last Updated on STN: 23 Oct 2003

AB Autism is a disorder characterised by severe difficulties in social interaction and communication, and with unusual behaviours. Once thought of as rare, autism is now recognised as being common. The role of CNS factors in pathogenesis is suggested by high rates of seizure disorder; research has highlighted the role of several specific brain regions in syndrome pathogenesis. Autism is a strongly genetic disorder and probably arises because of multiple genes; recurrence rates in families with one child are high. Early intervention with various techniques is helpful in many cases. Some pharmacological agents may help with certain problematic behaviours but do not address the underlying cause of the disorder.

L68 ANSWER 13 OF 30 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 3

ACCESSION NUMBER: 2003:605851 CAPLUS
DOCUMENT NUMBER: 140:71310
TITLE: Secretin activates visceral brain regions in the rat including areas abnormal in autism
AUTHOR(S): Welch, Martha G.; Keune, Jason D.; Welch-Horan, T. Bramwell; Anwar, Nargis; Anwar, Muhammad; Ruggiero, David A.
CORPORATE SOURCE: Division of Neuroscience, Laboratories of Childhood Regulatory Disorders and Behavioral Neuroanatomy, Riverside Drive, NYSPI, Columbia University College of Physicians and Surgeons, Department of Psychiatry, Columbia University College of Physicians and Surgeons, New York, NY, USA
SOURCE: Cellular and Molecular Neurobiology (2003), 23(4/5), 817-837
CODEN: CMNEDI; ISSN: 0272-4340
PUBLISHER: Kluwer Academic/Plenum Publishers
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The aim of this study was to determine whether central networks are involved in

the presumptive behavioral and autonomic regulatory actions of secretin, a gut hormone that has been reported to have ameliorative effects in autistic children. Central neural responses monitored by regional c-fos gene expression were examined in response to intracerebroventricular secretin injection in awake, freely-moving Sprague-Dawley rats. Tissue sections were incubated in an antibody to the c-fos gene product, Fos, and processed immunohistochem. Qual. differences in Fos immunoreactivity in stress adaptation and visceral representation areas of the brain were observed between secretin- and vehicle-infused age-matched pairs. Secretin-activated regions include the area postrema, dorsal motor nucleus, medial region of the nucleus of the solitary tract and its relay station in the lateral tegmentum, locus ceruleus, ventral periaqueductal gray, periventricular thalamic nucleus, paraventricular hypothalamus magnocellularis, medial and central amygdala, lateral septal complex as well as ependymal and subependymal nuclei lining the third ventricle. Specific areas of the cerebral cortex were heavily labeled in secretin-treated rats, as compared to controls: the medial bank of the anterior prefrontal cortex, orbitofrontal cortex, the piriform cortex, and the anterior olfactory nucleus. Secretin attenuated Fos immunoreactivity in the dorsal periaqueductal gray, intralaminar thalamus, medial parvicellular compartment of the hypothalamus, supraoptic nucleus of the hypothalamus, lateral amygdala, motor cortex, and the somatosensory and association areas of the parietal cortex. Secretin alters the activity of structures involved in behavioral conditioning of stress adaptation and visceral reflex reactions. This study predicts a possible cellular mechanism, activation of third ventricular ependymal and subependymal cells, as well as central regulatory actions of secretin. The physiol. effects of secretin on behavioral, endocrine, autonomic and sensory neuronal activation patterns, together, contribute to central c-fos activation. Secretin alters the activity of structures involved in behavioral conditioning of stress adaptation and visceral reflex reactions. This study predicts a possible cellular mechanism, activation of third ventricular ependymal and subependymal cells, and central regulatory actions of secretin. The physiol. effects of secretin on behavioral, endocrine, autonomic and sensory neuronal activation patterns, together, contribute to central c-fos activation. These findings mandate further investigation of secretin as a brain/gut stress regulatory hormone.

REFERENCE COUNT: 123 THERE ARE 123 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L68 ANSWER 14 OF 30 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 4

ACCESSION NUMBER: 2003:306157 CAPLUS
DOCUMENT NUMBER: 139:34798
TITLE: Does the MMR vaccine and secretin or its receptor share an antigenic epitope?
AUTHOR(S): Mehta, Bijal K.; Munir, Kerim M.
CORPORATE SOURCE: Memorial University of Newfoundland, St. John's, NF, Can.
SOURCE: Medical Hypotheses (2003), 60(5), 650-653
CODEN: MEHYDY; ISSN: 0306-9877
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review and discussion. In a subgroup of children with autism -spectrum like conditions symptoms seem to appear as a regression (in normal development). It has been postulated that the onset of such autistic symptoms may involve an autoimmune response against the central nervous system and that the antigenic determinant could possibly be gastrointestinal in origin. It has been suggested that the presence of the measles virus and autistic enterocolitis demonstrates the possibility that the MMR triple vaccine may be mediating the inflammation

with possible production of antibodies against the virus containing vaccine. Such an antibody may share antigenic determinant to mols. found in the gut. The authors propose that this may be secretin or its receptor, found in the gut as well as in the central nervous system. The antibody response to the gut may also conceivably occur in the brain at a critical time in development. The modulation of development by secretin may be a static event possibly occurring at a specific time in early childhood development and if it involves an autoimmune response then a disruption in development may result. These hypothesized events can only occur if the MMR vaccine shares antigenic determinants that resemble secretin or any of its receptor types.

REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L68 ANSWER 15 OF 30 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2003386723 EMBASE
TITLE: Intestinal pathophysiology in autism.
AUTHOR: White J.F.
CORPORATE SOURCE: J.F. White, Department of Physiology, Emory University, Atlanta, GA 30322, United States. jfwhite@physio.emory.edu
SOURCE: Experimental Biology and Medicine, (2003) Vol. 228, No. 6, pp. 639-649. .
Refs: 98
ISSN: 1535-3702 CODEN: EBMMBE
COUNTRY: United States
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 032 Psychiatry
037 Drug Literature Index
038 Adverse Reactions Titles
039 Pharmacy
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 16 Oct 2003
Last Updated on STN: 16 Oct 2003

AB Autism is a life-long developmental disorder affecting as many as 1 in 500 children. The causes for this profound disorder are largely unknown. Recent research has uncovered pathology in the gastrointestinal tract of autistic children. The pathology, reported to extend from the esophagus to the colon, is described here along with other studies pointing to a connection between diet and the severity of symptoms expressed in autism. The evidence that there is impaired intestinal permeability in autism is reviewed, and various theories are discussed by which a leaky gut could develop. Lastly, some possible ways in which impaired gastrointestinal function might influence brain function are discussed.

L68 ANSWER 16 OF 30 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 5

ACCESSION NUMBER: 2004:44082 CAPLUS
DOCUMENT NUMBER: 140:216004
TITLE: Infections, toxic chemicals and dietary peptides binding to lymphocyte receptors and tissue enzymes are major instigators of autoimmunity in autism
AUTHOR(S): Vojdani, A.; Pangborn, J. B.; Vojdani, E.; Cooper, E. L.
CORPORATE SOURCE: Laboratory of Comparative Neuroimmunology, Department of Neurobiology, David Geffen School of Medicine at UCLA, University of California, Los Angeles, Los Angeles, CA, 90095, USA
SOURCE: International Journal of Immunopathology and Pharmacology (2003), 16(3), 189-199
CODEN: IJIPE4; ISSN: 0394-6320

PUBLISHER: Biolife s.a.s.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Similar to many complex autoimmune diseases, genetic and environmental factors including diet, infection and xenobiotics play a critical role in the development of autism. In this study, we postulated that infectious agent antigens such as streptokinase, dietary peptides (gliadin and casein) and Et mercury (xenobiotic) bind to different lymphocyte receptors and tissue enzyme (DPP IV or CD26 ). We assessed this hypothesis first by measuring IgG, IgM and IgA antibodies against CD26, CD69, streptokinase (SK), gliadin and casein peptides and against Et mercury bound to human serum albumin in patients with autism. A significant percentage of children with autism developed anti-SK, anti-gliadin and casein peptides and anti-Et mercury antibodies, concomitant with the appearance of anti-CD26 and anti-CD69 autoantibodies. These antibodies are synthesized as a result of SK, gliadin, casein and Et mercury binding to CD26 and CD69, indicating that they are specific. Immune absorption demonstrated that only specific antigens, like CD26, were capable of significantly reducing serum anti-CD26 levels. However, for direct demonstration of SK, gliadin, casein and Et mercury to CD26 or CD69, microtiter wells were coated with CD26 or CD69 alone or in combination with SK, gliadin, casein or Et mercury and then reacted with enzyme labeled rabbit anti-CD26 or anti-CD69. Adding these mols. to CD26 or CD69 resulted in 28-86% inhibition of CD26 or CD69 binding to anti-CD26 or anti-CD69 antibodies. The highest % binding of these antigens or peptides to CD26 or CD69 was attributed to SK and the lowest to casein peptides. We, therefore, propose that bacterial antigens (SK), dietary peptides (gliadin, casein) and Thimerosat (Et mercury) in individuals with pre-disposing HLA mols.; bind to CD26 or CD69 and induce antibodies against these mols. In conclusion, this study is apparently the first to demonstrate that dietary peptides, bacterial toxins and xenobiotics bind to lymphocyte receptors and/or tissue enzymes, resulting in autoimmune reaction in children with autism.
REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L68 ANSWER 17 OF 30 MEDLINE on STN
ACCESSION NUMBER: 2003067646 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12578238
TITLE: Opioid peptides and dipeptidyl peptidase in autism

AUTHOR: Hunter L C; O'Hare A; Herron W J; Fisher L A; Jones G E
CORPORATE SOURCE: YAbA Ltd, Logan Building, Roslin Biocentre, Midlothian, UK.
SOURCE: Developmental medicine and child neurology, (2003 Feb) Vol. 45, No. 2, pp. 121-8.
Journal code: 0006761. ISSN: 0012-1622.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200302
ENTRY DATE: Entered STN: 12 Feb 2003
Last Updated on STN: 27 Feb 2003
Entered Medline: 26 Feb 2003
AB It has been hypothesized that autism results from an 'opioid peptide excess'. The aims of this study were to (1) confirm the presence of opioid peptides in the urine of children with autism and (2) determine whether dipeptidyl peptidase IV (DPPIV/CD26) is defective in children with autism.
Opioid peptides were not detected in either the urine of children with

autism (10 children; nine males, one female; age range 2 years 6 months to 10 years 1 month) or their siblings (10 children; seven males, three females; age range 2 years 3 months to 12 years 7 months) using liquid chromatography-ultraviolet-mass spectrometric analysis (LC-UV-MS). Plasma from 11 normally developing adults (25 years 5 months to 55 years 5 months) was also tested. The amount and activity of DPPIV in the plasma were quantified by an ELISA and DPPIV enzyme assay respectively; DPPIV was not found to be defective. The percentage of mononuclear cells expressing DPPIV (as CD26) was determined by flow cytometry. Children with autism had a significantly lower percentage of cells expressing CD3 and CD26, suggesting that they had lower T-cell numbers than their siblings. In conclusion, this study failed to replicate the findings of others and questions the validity of the opioid peptide excess theory for the cause of autism.

L68 ANSWER 18 OF 30 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 2004:197426 BIOSIS
DOCUMENT NUMBER: PREV200400197985
TITLE: Distribution, specificity of secretin in brain: neuroregulatory role in autism.
AUTHOR(S): Ruggiero, D. A. [Reprint Author]; Welch-Horan, T. B.; Keune, J. D.; Anwar, N.; Anwar, M.; Ludwig, R. J.; Welch, M. G.
CORPORATE SOURCE: Psychiatry/Neurosci., Columbia Univ. Col. Physicians and Surgeons, NY, NY, USA
SOURCE: Society for Neuroscience Abstract Viewer and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. 318.6. http://sfn.scholarone.com. e-file.
Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 08-12, 2003. Society of Neuroscience.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Apr 2004
Last Updated on STN: 14 Apr 2004

AB Aim: To resolve secretin synthesis by brain/gut stress axes and neuronal mechanisms underlying secretin's actions in treating autism. Method: A sensitive immunocytochemical technique with single cell resolution was performed in ten age/weight-matched male rats pretreated by stereotaxic intracerebroventricular microinjection of colchicine (0.6 micrograms/kg) or vehicle. Following 2-day survival, rats were anesthetized and perfused for immunocytochemistry. Forebrain and intestinal segments were blocked and alternate frozen 30 micron sections incubated in polyclonal antibodies against secretin, vasoactive intestinal peptide, glucagon or PACAP. Sections through brain and gut were processed for Nissl or HEPSILON stains. Preadsorption control studies were performed with members of the secretin peptide family to demonstrate specificity. Results: Specificity of secretin immunoreactivity(ir) was verified by clear-cut preadsorption control data and relatively high concentrations and distinct topographic localization of secretin-ir to paraventricular/supraoptic and intercalated hypothalamic nuclei and upper intestinal tract. Secretin levels were up-regulated by colchicine, an exemplar of homeostatic stressors, as compared with low constitutive expression in untreated rats. CONCLUSION: This study provides the first direct immunocytochemical demonstration of secretin-ir in the forebrain. The hypothalamus, like the gut, is capable of synthesizing secretin, pointing to dual peripheral/central roles for secretin in conditioning stress adaptation. Secretin expression by brain and gut is stress-related and, as suggested by the distribution patterns, may interact with other stress-adaptation hormones. Secretin may be

synthesized on demand in response to homeostatic challenges, explaining its mechanisms of action in autism.

L68 ANSWER 19 OF 30 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:906554 CAPLUS
DOCUMENT NUMBER: 138:1044
TITLE: G protein-coupled receptor (GPCR) microarrays for determination of GPCR gene expression profiles and uses in drug and toxin screening and diagnostics
INVENTOR(S): Thirstrup, Kenneth; Madsen, Lars Siim; Jensen, Jens Bitsch; Hummel, Rene; Jensen, Bo Skaaning
PATENT ASSIGNEE(S): Azign Bioscience A/s, Den.
SOURCE: PCT Int. Appl., 43 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2002095065 | A2 | 20021128 | WO 2002-DK337 | 20020521 |
| WO 2002095065 | A3 | 20040325 | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW | | | | |
| RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| EP 1423530 | A2 | 20040602 | EP 2002-724150 | 20020521 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR | | | | |
| US 2004171008 | A1 | 20040902 | US 2003-477399 | 20031112 |
| PRIORITY APPLN. INFO.: | | | DK 2001-802 A | 20010518 |
| | | | WO 2002-DK337 W | 20020521 |

AB The invention provides G protein-coupled receptor (GPCR) arrays, kits comprising GPCR arrays and methods to produce such GPCR arrays. GPCR arrays are useful in the determination of GPCR expression profiles in biol. materials and also in the identification of therapeutic, prophylactic and/or toxic agents involved in the response of the GPCR expression. The invention relates to an GPCR array comprising a multiplicity of individual GPCR polynucleotide spots stably associated with a surface of a solid support, wherein an individual GPCR polynucleotide spot comprises an GPCR polynucleotide composition comprising a non-conserved region of an GPCR polynucleotide family member, the spots representing at least two different regions of an GPCR polynucleotide member of a family. The invention also relates to a set of primers specific for nonconserved regions of GPCR polynucleotide family members, wherein the set of primers are used in the method for the production of an array according to the invention. In still a further aspect, the invention relates to a diagnostic method to determine the differences of GPCR expression profiles between two different biol. materials.

L68 ANSWER 20 OF 30 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:276155 CAPLUS
DOCUMENT NUMBER: 136:305198
TITLE: Sequences of a novel human secretin receptor-like G protein-coupled receptor sequence homolog and uses in diagnosis, therapy and drug screening

INVENTOR(S): Liou, Jiing-Ren
PATENT ASSIGNEE(S): Bayer Aktiengesellschaft, Germany
SOURCE: PCT Int. Appl., 122 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | | DATE |
|---|---|---|---|---|---|
| WO 2002029052 | A2 | 20020411 | WO 2001-EP11515 | | 20011005 |
| WO 2002029052 | A3 | 20021227 | | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM | | | | | |
| RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | | |
| AU 2002010522 | A5 | 20020415 | AU 2002-10522 | | 20011005 |
| EP 1326975 | A2 | 20030716 | EP 2001-978398 | | 20011005 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR | | | | | |
| US 2004048273 | A1 | 20040311 | US 2003-398454 | | 20031008 |
| PRIORITY APPLN. INFO.: | | | US 2000-238125P | P | 20001006 |
| | | | US 2001-261756P | P | 20010117 |
| | | | WO 2001-EP11515 | W | 20011005 |

AB The invention provides protein and cDNA sequences of a novel human secretin receptor-like G protein-coupled receptor sequence homolog. The invention also provides reagents and methods of regulating a human secretin receptor-like G protein-coupled receptor sequence homolog. Reagents which regulate human secretin receptor receptor-like GPCR and reagents which bind to human secretin receptor-like GPCR gene products can play a role in preventing, ameliorating, or correcting dysfunctions or diseases including, but not limited to, cardiovascular disorders, urinary incontinence, benign prostate hyperplasia, obesity and diseases related to obesity, cancer, diabetes, osteoporosis, anxiety, depression, hypertension, migraine, compulsive disorders, schizophrenia, autism, neurodegenerative disorders, such as Alzheimer's disease, Parkinsonism, and Huntington's chorea, and cancer chemotherapy-induced vomiting.

L68 ANSWER 21 OF 30 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:276154 CAPLUS
DOCUMENT NUMBER: 136:305197
TITLE: Sequences of a novel human secretin receptor-like G protein-coupled receptor sequence homolog and uses in diagnosis, therapy and drug screening
INVENTOR(S): Liou, Jiing-Ren
PATENT ASSIGNEE(S): Bayer Aktiengesellschaft, Germany
SOURCE: PCT Int. Appl., 113 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2002029051 | A2 | 20020411 | WO 2001-EP11443 | 20011004 |

WO 2002029051 A3 20030320
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
AU 2002018205 A5 20020415 AU 2002-18205 20011004
EP 1326977 A2 20030716 EP 2001-986319 20011004
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
US 2004058350 A1 20040325 US 2003-398455 20030923
PRIORITY APPLN. INFO.: US 2000-238043P P 20001006
WO 2001-EP11443 W 20011004

AB The invention provides protein and cDNA sequences of a novel human secretin receptor-like G protein-coupled receptor sequence homolog. The invention also provides reagents and methods of regulating a human secretin receptor-like G protein-coupled receptor sequence homolog. Reagents which regulate human secretin receptor receptor-like GPCR and reagents which bind to human secretin receptor-like GPCR gene products can play a role in preventing, ameliorating, or correcting dysfunctions or diseases including, but not limited to, cardiovascular disorders, urinary incontinence, benign prostate hyperplasia, obesity and diseases related to obesity, cancer, diabetes, osteoporosis, anxiety, depression, hypertension, migraine, compulsive disorders, schizophrenia, autism, neurodegenerative disorders, such as Alzheimer's disease, Parkinsonism, and Huntington's chorea, and cancer chemotherapy-induced vomiting.

L68 ANSWER 22 OF 30 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:276153 CAPLUS
DOCUMENT NUMBER: 136:305196
TITLE: Sequences of a novel human secretin receptor-like G protein-coupled receptor sequence homolog and uses in diagnosis, therapy and drug screening
INVENTOR(S): Liou, Jiing-Ren
PATENT ASSIGNEE(S): Bayer Aktiengesellschaft, Germany
SOURCE: PCT Int. Appl., 133 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2002029050 | A2 | 20020411 | WO 2001-EP11442 | 20011004 |
| WO 2002029050 | A3 | 20030522 | | |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW | | | | |
| RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| AU 2002012309 | A5 | 20020415 | AU 2002-12309 | 20011004 |
| EP 1328637 | A2 | 20030723 | EP 2001-980476 | 20011004 |

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

US 2004096847 A1 20040520 US 2003-398448 20030404
PRIORITY APPLN. INFO.: US 2000-238045P P 20001006
US 2001-315958P P 20010831
WO 2001-EP11442 W 20011004

AB The invention provides protein and cDNA sequences of a novel human secretin receptor-like G protein-coupled receptor sequence homolog. The invention also provides reagents and methods of regulating a human secretin receptor-like G protein-coupled receptor sequence homolog. Reagents which regulate human secretin receptor receptor-like GPCR and reagents which bind to human secretin receptor-like GPCR gene products can play a role in preventing, ameliorating, or correcting dysfunctions or diseases including, but not limited to, cardiovascular disorders, urinary incontinence, benign prostate hyperplasia, obesity and diseases related to obesity, cancer, diabetes, osteoporosis, anxiety, depression, hypertension, migraine, compulsive disorders, schizophrenia, autism, neurodegenerative disorders, such as Alzheimer's disease, Parkinsonism, and Huntington's chorea, and cancer chemotherapy-induced vomiting.

L68 ANSWER 23 OF 30 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:276025 CAPLUS
DOCUMENT NUMBER: 136:304110
TITLE: Regulation of human secretin receptor-like GPCR
INVENTOR(S): Kossida, Sophia
PATENT ASSIGNEE(S): Bayer Aktiengesellschaft, Germany
SOURCE: PCT Int. Appl., 125 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2002028898 | A2 | 20020411 | WO 2001-EP11439 | 20011004 |
| WO 2002028898 | A3 | 20030605 | | |

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

| | | | | |
|---|---|---|---|---|
| AU 2002012308 | A5 | 20020415 | AU 2002-12308 | 20011004 |
| EP 1332215 | A2 | 20030806 | EP 2001-980475 | 20011004 |

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

US 2004024184 A1 20040205 US 2003-398458 20030717
PRIORITY APPLN. INFO.: US 2000-238126P P 20001006
WO 2001-EP11439 W 20011004

AB Reagents which regulate human secretin receptor-like GPCR and reagents which bind to human secretin-like GPCR gene products can play a role in preventing, ameliorating, or correcting dysfunctions or diseases including, but not limited to, obesity and diseases related to obesity, cancer, diabetes, osteoporosis, anxiety, depression, hypertension, migraine, compulsive disorders, schizophrenia, autism, neurodegenerative disorders, such as Alzheimer's disease, Parkinsonism, and Huntington's chorea, and cancer chemotherapy-induced

vomiting.

L68 ANSWER 24 OF 30 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2002228620 EMBASE
TITLE: MMR vaccination, ileal lymphoid nodular hyperplasia, and pervasive developmental disorder.
AUTHOR: Hendrickson B.A.; Turner J.R.
CORPORATE SOURCE: B.A. Hendrickson, Section of Pediatric Infectious Dis., Department of Pathology, University of Chicago, Chicago, IL 60637, United States. bhendric@peds.bsd.uchicago.edu
SOURCE: Lancet, (15 Jun 2002) Vol. 359, No. 9323, pp. 2051-2052. .
Refs: 17
ISSN: 0140-6736 CODEN: LANCAO
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Note
FILE SEGMENT: 006 Internal Medicine
007 Pediatrics and Pediatric Surgery
026 Immunology, Serology and Transplantation
037 Drug Literature Index
048 Gastroenterology
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Jul 2002
Last Updated on STN: 11 Jul 2002
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L68 ANSWER 25 OF 30 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2002337157 EMBASE
TITLE: Autistic disorder and gastrointestinal disease.
AUTHOR: Horvath K.; Perman J.A.
CORPORATE SOURCE: Dr. K. Horvath, Div. of Pediatric Gastroenterology, Univ. of Maryland School of Medicine, Box 140, 22 South Greene Street, Baltimore, MD 21201-1595, United States. khorvath@pol.net
SOURCE: Current Opinion in Pediatrics, (2002) Vol. 14, No. 5, pp. 583-587. .
Refs: 25
ISSN: 1040-8703 CODEN: COPEE
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 007 Pediatrics and Pediatric Surgery
029 Clinical Biochemistry
032 Psychiatry
037 Drug Literature Index
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 3 Oct 2002
Last Updated on STN: 3 Oct 2002

AB Autistic disorder is a pervasive developmental disorder manifested in the first 3 years of life by dysfunction in social interaction and communication. Many efforts have been made to explore the biologic basis of this disorder, but the etiology remains unknown. Recent publications describing upper gastrointestinal abnormalities and ileocolitis have focused attention on gastrointestinal function and morphology in these children. High prevalence of histologic abnormalities in the esophagus, stomach, small intestine and colon, and dysfunction of liver conjugation capacity and intestinal permeability were reported. Three surveys conducted in the United States described high prevalence of gastrointestinal symptoms in children with autistic disorder. Treatment of the digestive problems may have positive effects on their behavior. .COPYRGT. 2002 Lippincott Williams & Wilkins, Inc.

L68 ANSWER 26 OF 30 MEDLINE on STN
ACCESSION NUMBER: 2002315738 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12056881
TITLE: Enzyme-based therapy for autism spectrum disorders -- is it worth another look?.
AUTHOR: Brudnak Mark A; Rimland Bernard; Kerry Roy E; Dailey Margaret; Taylor Robert; Stayton Bruce; Waickman Frank; Waickman Michael; Pangborn Jon; Buchholz Ilene
CORPORATE SOURCE: Autism Research Institute, San Diego, CA, USA.
SOURCE: Medical hypotheses, (2002 May) Vol. 58, No. 5, pp. 422-8. Journal code: 7505668. ISSN: 0306-9877.
PUB. COUNTRY: Scotland: United Kingdom
DOCUMENT TYPE: (CLINICAL TRIAL) Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200302
ENTRY DATE: Entered STN: 12 Jun 2002
Last Updated on STN: 7 Feb 2003
Entered Medline: 6 Feb 2003

AB Autism is a developmental disease usually manifesting within the first three years of life. To date, no causative agent has been found. Similarly, treatment options have been limited. Of the treatment options available, a number of them have been nutritionally based in an attempt to address one or more of the theories regarding the etiology of the disease. An example would be enzyme therapy for the digestion of purported offending neuroactive peptides collectively known as exorphins. This paper discusses the exorphin theory of autism and subsequent treatment with dietary enzyme therapy. Novel data are presented in support of the theory that enzymes play a critical role in autism . Forty-six patients between the ages of 5 and 31 were selected for inclusion in the study based on a diagnosis placing them in the category of the autism spectrum disorders (ASD). The diets were supplemented with a novel dietary enzyme formulation, ENZYMAID, for a period of 12 weeks. Progress was tracked according to the Symptom Outcome Survey (SOS) (1) form method of symptom charting and presented in a table for further analysis. The novel enzyme formula, ENZYMAID, beneficially and safely affected all 13 of the parameters measured. Improvements ranged from 50-90%, depending on the parameter measured. Enzyme therapy to treat ASD may indeed a viable option in treatment protocols. These results indicate that further controlled studies are warranted.


L68 ANSWER 27 OF 30 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2002:895643 CAPLUS
DOCUMENT NUMBER: 138:168045
TITLE: Effects of neonatal 6-hydroxydopamine lesion on the gene expression profile in young adult rats
AUTHOR(S): Masuo, Yoshinori; Ishido, Masami; Morita, Masatoshi; Oka, Syuichi
CORPORATE SOURCE: International Patent Organism Depositary, National Institute of Advanced Industrial Science and Technology (AIST), Tsukuba, Ibaraki, 305-8566, Japan
SOURCE: Neuroscience Letters (2002), 335(2), 124-128
CODEN: NELED5; ISSN: 0304-3940
PUBLISHER: Elsevier Science Ireland Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Patients with pervasive developmental disorders, including autism , and attention-deficit hyperactivity disorder show behavioral hyperactivity during childhood. We investigated the effects of a neonatal 6-hydroxydopamine lesion on multiple gene expression in the rat striatum and midbrain. Spontaneous motor activity was significantly increased at 4-5 wk of age. The animals were sacrificed, and the striatum and midbrain

were subjected to gene expression profiling using a membrane array with 1176 kinds of cDNAs. Alterations were found in several classes of gene expression, depending on the brain region. Enhanced expression of the glutamate transporter gene was found in the striatum. Expression of the dopamine receptor D4 gene and dopamine transporter gene was also increased in the midbrain. These results suggest that 6-hydroxydopamine-treated rats may partly mimic human hyperkinesia not only in behavior but also in gene expression.

REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L68 ANSWER 28 OF 30 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 6

ACCESSION NUMBER: 2003:381142 BIOSIS
DOCUMENT NUMBER: PREV200300381142
TITLE: SECRETIN ACTIVATES VISCERAL BRAIN REGIONS INCLUDING AREAS ABNORMAL IN AUTISM.
AUTHOR(S): Welch, M. G. [Reprint Author]; Keune, J. D. [Reprint Author]; Welch-Horan, T. B. [Reprint Author]; Ruggiero, D. A. [Reprint Author]
CORPORATE SOURCE: Psychiatry, Anatomy, Columbia University College of Physicians and Surgeons, New York, NY, USA
SOURCE: Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002) Vol. 2002, pp. Abstract No. 896.2. http://sfn.scholarone.com. cd-rom.
Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience. Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 20 Aug 2003
Last Updated on STN: 20 Aug 2003

AB Secretin, a 27 amino acid gut/brain neuropeptide, has reported behavioral effects in autistic children. We examined regional Fos immunoreactivity in response to i.c.v. secretin (10-30mug) injection in sentient S.D. rats. Tissue sections were processed immunohistochemically with c-fos specific antibody and examined three hours later. Qualitative differences in Fos immunoreactivity were observed between secretin-and vehicle-infused age-matched pairs (n=4 pairs). Secretin-infused rats showed altered numbers of Fos-immunoreactive nuclei in visceral representation areas of the brain. Secretin-activated regions include the area postrema, medial region of the nucleus of the solitary tract and its relay station in the lateral tegmentum, ventral periaqueductal gray, periventricular thalamic nucleus, paraventricular hypothalamus magnocellularis, medial and cortical amygdala, piriform cortex, lateral septal complex, medial bank of the anterior prefrontal cortex as well as ependymal and subependymal nuclei. Secretin attenuated Fos immunoreactivity in the dorsal periaqueductal gray, the intralaminar thalamus, the medial parvicellular, CRH, compartment of the hypothalamus, and the somatosensory and association areas of the parietal cortex. The regions effected by secretin in this study overlap with regions identified as being abnormal in autistic brains. Conclusions. Secretin alters the activity of structures involved in behavioral conditioning of stress adaptation reactions and visceral reflex adjustments. This study provides evidence for the regulatory actions of secretin and its possible therapeutic use in autism.

L68 ANSWER 29 OF 30 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 2000:499731 BIOSIS
DOCUMENT NUMBER: PREV200000499852

TITLE: Safe use of intravenous secretin in autistic children.
AUTHOR(S): Sockolow, Robbyn [Reprint author]; Meckes, David [Reprint author]; Hewitson, Kerri [Reprint author]; Atluru, Vijaya [Reprint author]
CORPORATE SOURCE: Pediatrics, Winthrop-University Hosp., Mineola, NY, USA
SOURCE: JPGN, (2000) Vol. 31, No. Supplement 2, pp. S156. print. Meeting Info.: World Congress of Pediatric Gastroenterology, Hepatology, and Nutrition. Boston, Massachusetts, USA. August 05-09, 2000.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 15 Nov 2000
Last Updated on STN: 10 Jan 2002

L68 ANSWER 30 OF 30 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2000:482368 BIOSIS
DOCUMENT NUMBER: PREV200000482368
TITLE: Evaluation of gastrointestinal symptoms in autistic children before and following secretin infusion.
AUTHOR(S): Lightdale, J. R. [Reprint author]; Hayer, C.; Siegel, B.; Elliott, G. R.; Heyman, M. B.
CORPORATE SOURCE: Gastroenterology and Nutrition, Children's Hospital, Boston, MA, USA
SOURCE: JPGN, (2000) Vol. 31, No. Supplement 2, pp. S31. print. Meeting Info.: World Congress of Pediatric Gastroenterology, Hepatology, and Nutrition. Boston, Massachusetts, USA. August 05-09, 2000.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 8 Nov 2000
Last Updated on STN: 10 Jan 2002

=> (CD13, CD26, transglutaminase, DPPI, dipeptidyl peptidase IV, secretin, gastrin, motilin) and (autism or autistic) and (antigen or antibody)
L69 0 FILE CAPLUS
L70 0 FILE BIOSIS
L71 0 FILE MEDLINE
L72 0 FILE EMBASE
L73 0 FILE USPATFULL

TOTAL FOR ALL FILES
L74 0 (CD13, CD26, TRANSGLUTAMINASE, DPPI, DIPEPTIDYL PEPTIDASE IV, SECRETIN, GASTRIN, MOTILIN) AND (AUTISM OR AUTISTIC) AND (ANTIGEN OR ANTIBODY)

result in loss of user privileges and other penalties.

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FILE 'HOME' ENTERED AT 14:27:48 ON 24 JAN 2007

=> file .meeting'
'.MEETING'' IS NOT A VALID FILE NAME
SESSION CONTINUES IN FILE 'HOME'
Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files
that are available. If you have requested multiple files, you can
specify a corrected file name or you can enter "IGNORE" to continue
accessing the remaining file names entered.

=> file .meeting
'EVENTLINE' IS NOT A VALID FILE NAME
Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files
that are available. If you have requested multiple files, you can
specify a corrected file name or you can enter "IGNORE" to continue
accessing the remaining file names entered.
ENTER A FILE NAME OR (IGNORE):ignore
'MEDICONF' IS NOT A VALID FILE NAME
Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files
that are available. If you have requested multiple files, you can
specify a corrected file name or you can enter "IGNORE" to continue
accessing the remaining file names entered.
ENTER A FILE NAME OR (IGNORE):ignore
COST IN U.S. DOLLARS

| | SINCE FILE ENTRY | TOTAL SESSION |
|---|---|---|
| FULL ESTIMATED COST | 0.42 | 0.42 |

FILE 'AGRICOLA' ENTERED AT 14:28:56 ON 24 JAN 2007

FILE 'BIOTECHNO' ENTERED AT 14:28:56 ON 24 JAN 2007
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FILE 'HEALSAFE' ENTERED AT 14:28:56 ON 24 JAN 2007
COPYRIGHT (C) 2007 Cambridge Scientific Abstracts (CSA)

FILE 'IMSDRUGCONF' ENTERED AT 14:28:56 ON 24 JAN 2007
COPYRIGHT (C) 2007 IMSWORLD Publications Ltd.

FILE 'LIFESCI' ENTERED AT 14:28:56 ON 24 JAN 2007
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FILE 'PASCAL' ENTERED AT 14:28:56 ON 24 JAN 2007
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=> (autism or autistic) and (measls and mumps and rubella)
L1 0 FILE AGRICOLA
L2 0 FILE BIOTECHNO
L3 0 FILE CONFSCI
L4 0 FILE HEALSAFE
L5 0 FILE IMSDRUGCONF
L6 0 FILE LIFESCI
L7 0 FILE PASCAL

```
TOTAL FOR ALL FILES
L8            0 (AUTISM OR AUTISTIC) AND (MEASLS AND MUMPS AND RUBELLA)

=> (autistic or autism) and meals and mumps and rubella
L9            0 FILE AGRICOLA
L10           0 FILE BIOTECHNO
L11           0 FILE CONFSCI
L12           0 FILE HEALSAFE
L13           0 FILE IMSDRUGCONF
L14           0 FILE LIFESCI
L15           0 FILE PASCAL

TOTAL FOR ALL FILES
L16           0 (AUTISTIC OR AUTISM) AND MEALS AND MUMPS AND RUBELLA

=> meals and mumps and rubella and (autism or autistic)
L17           0 FILE AGRICOLA
L18           0 FILE BIOTECHNO
L19           0 FILE CONFSCI
L20           0 FILE HEALSAFE
L21           0 FILE IMSDRUGCONF
L22           0 FILE LIFESCI
L23           0 FILE PASCAL

TOTAL FOR ALL FILES
L24           0 MEALS AND MUMPS AND RUBELLA AND (AUTISM OR AUTISTIC)

=>  measles and mumps and rubella and (autism or autistic)
L25           0 FILE AGRICOLA
L26          14 FILE BIOTECHNO
L27           0 FILE CONFSCI
L28          11 FILE HEALSAFE
L29           0 FILE IMSDRUGCONF
L30          25 FILE LIFESCI
L31          48 FILE PASCAL

TOTAL FOR ALL FILES
L32          98 MEASLES AND MUMPS AND RUBELLA AND (AUTISM OR AUTISTIC)

=> dup rem
ENTER L# LIST OR (END):l26
PROCESSING COMPLETED FOR L26
L33           14 DUP REM L26 (0 DUPLICATES REMOVED)

=> d l33 ibib abs total

L33   ANSWER 1 OF 14  BIOTECHNO  COPYRIGHT 2007 Elsevier Science B.V. on STN
ACCESSION NUMBER:         2003:37329864   BIOTECHNO
TITLE:                    Public opponents of vaccination: A case study
AUTHOR:                   Leask J.; McIntyre P.
CORPORATE SOURCE:         J. Leask, Natl. Ctr. Immunisation Res./S., Children's
                          Hospital at Westmead, University of Sydney, Locked Bag
                          4001, Westmead, NSW 2145, Australia.
                          E-mail: juliel3@chw.edu.au
SOURCE:                   Vaccine, (01 DEC 2003), 21/32 (4700-4703), 28
                          reference(s)
                          CODEN: VACCDE  ISSN: 0264-410X
DOCUMENT TYPE:            Journal; Article
COUNTRY:                  United Kingdom
LANGUAGE:                 English
SUMMARY LANGUAGE:         English
AN    2003:37329864   BIOTECHNO
AB    Opposition to mass childhood vaccination is a world-wide phenomenon,
```

particularly in industrialised countries. Unfounded claims about vaccination are perpetuated by parental lobby groups and individual spokespeople, some of whom have a medical or scientific background. This article focuses on one such spokesperson who has achieved particular notoriety. Dr. Viera Scheibner is a retired micropalaeontologist, without any formal training in health-related sciences, who tours the world claiming that vaccines are ineffective and dangerous and lead to a host of ills such as cancer and asthma. Professionals in public health or the clinical arena are from time to time called upon to publicly respond to her, or similar, claims disseminated during tours of Europe, North America or Australasia and in books and articles. Health professionals have expressed at how such spokespersons misrepresent the evidence on vaccine safety, resulting in the potential to undermine public confidence in immunisation. Media coverage, or proposed coverage, particularly of her more extreme claims, often makes health professionals engaged in immunisation feel obliged to respond. This paper describes Viera Scheibner's approach, which follows a repetitious path and is representative of that taken by other public opponents of immunisation. We conclude by suggesting how health professionals might respond in the public arena. 

L33 ANSWER 2 OF 14 BIOTECHNO 
ACCESSION NUMBER: 2003:37338229 BIOTECHNO
TITLE: The pertussis vaccine controversy in Great Britain, 1974-1986
AUTHOR: Baker J.P.
CORPORATE SOURCE: J.P. Baker, Ctr. Stud. of Med. Ethics/Hum., Duke University, Box 3040 DUMC, Durham, NC, United States. E-mail: baker009@mc.duke.edu
SOURCE: Vaccine, (2003), 21/25-26 (4003-4010), 52 reference(s) CODEN: VACCDE ISSN: 0264-410X
DOCUMENT TYPE: Journal; General Review
COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2003:37338229 BIOTECHNO
AB This historical essay analyzes the role played by Great Britain in the pertussis vaccine controversy of the 1970s and 1980s. Public backlash against this vaccine not only took place earlier in Britain than the United States, but also was so widespread that a series of whooping cough epidemics soon followed. As with the more recent dispute involving measles-mumps-rubella (MMR) vaccine and autism, the United Kingdom played a primary role in defining, promoting, and ultimately exporting this controversy. This essay seeks to explain this phenomenon by situating it in Britain's long history of suspicion regarding vaccines evident among both the public and the medical profession, a theme dating back to the compulsory vaccination laws of the 19th century. It argues that anti-vaccinationism, far from being simply a new development related to the public's lack of awareness of childhood vaccine-preventable illness, actually represents a revival of a much older movement.

L33 ANSWER 3 OF 14 BIOTECHNO 
ACCESSION NUMBER: 2003:37338223 BIOTECHNO
TITLE: Unintended events following immunization with MMR: A systematic review
AUTHOR: Jefferson T.; Price D.; Demicheli V.; Bianco E.
CORPORATE SOURCE: T. Jefferson, Reparto Epidemiologia Clinica, Istituto Superiore di Sanita, Viale Regina Elena, 299-00161 Rome, Italy. E-mail: toj1@aol.com
SOURCE: Vaccine, (2003), 21/25-26 (3954-3960), 35 reference(s) CODEN: VACCDE ISSN: 0264-410X
DOCUMENT TYPE: Journal; General Review

COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2003:37338223 BIOTECHNO
AB Public debate over the safety of the trivalent measles, mumps and rubella (MMR) vaccine and the drop in vaccination rates in several countries persists despite its almost universal use and accepted effectiveness. We carried out a systematic review to assess the evidence of unintended effects (beneficial or harmful) associated with MMR and the applicability of systematic reviewing methods to the field of safety evaluation. Eligible studies were comparative prospective or retrospective on healthy individuals up to 15 years of age, carried out or published by 2003. We identified 120 articles satisfying our inclusion criteria and included 22. MMR is associated with a lower incidence of upper respiratory tract infections, a higher incidence of irritability, similar incidence of other adverse effects compared to placebo and is likely to be associated with benign thrombocytopenic purpura (TP), parotitis, joint and limb complaints and aseptic meningitis (mumps Urabe strain-containing MMR). Exposure to MMR is unlikely to be associated with Crohn's disease, ulcerative colitis, autism or aseptic meningitis (mumps Jeryl-Lynn strain-containing MMR). The design and reporting of safety outcomes in MMR vaccine studies, both pre- and post-marketing, are largely inadequate. The evidence of adverse events following immunization with MMR cannot be separated from its role in preventing the target diseases. .COPYRGT. 2003 Elsevier Ltd. All rights reserved.

L33 ANSWER 4 OF 14 BIOTECHNO COPYRIGHT 2007 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2003:36950051 BIOTECHNO
TITLE: Lipid and carbohydrate based adjuvant/carriers in immunology
AUTHOR: McGeary R.P.; Olive C.; Toth I.
CORPORATE SOURCE: I. Toth, School of Molec./Microbial Sciences, School of Pharmacy, The University of Queensland, Brisbane, QLD 4072, Australia.
E-mail: i.toth@pharmacy.uq.edu.au
SOURCE: Journal of Peptide Science, (01 JUL 2003), 9/7 (405-418), 96 reference(s)
CODEN: JPSIEI ISSN: 1075-2617
DOCUMENT TYPE: Journal; General Review
COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2003:36950051 BIOTECHNO
AB This review discusses various issues regarding vaccines; what are they and how they work, safety aspects, the role of adjuvants and carriers in vaccination, synthetic peptides as immunogens, and new technologies for vaccine development and delivery including the identification of novel adjuvants for mucosal vaccine delivery. There has been a recent increase of interest in the use of lipids and carbohydrates as adjuvants, and so a particular emphasis is placed on adjuvants derived from lipids or carbohydrates, or from both. Copyright .COPYRGT. 2003 European Peptide Society and John Wiley & Sons, Ltd.

L33 ANSWER 5 OF 14 BIOTECHNO COPYRIGHT 2007 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2003:37392947 BIOTECHNO
TITLE: Measles Virus 1998-2002: Progress and Controversy
AUTHOR: Rall G.F.
CORPORATE SOURCE: G.F. Rall, Division of Basic Science, Fox Chase Cancer Center, 7701 Burholme Avenue, Philadelphia, PA 19111, United States.
E-mail: gf_rall@fccc.edu
SOURCE: Annual Review of Microbiology, (2003), 57/- (343-367),

133 reference(s)
CODEN: ARMIAZ ISSN: 0066-4227
DOCUMENT TYPE: Journal; General Review
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2003:37392947 BIOTECHNO
AB Despite the extensive media exposure that viruses such as West Nile, Norwalk, and Ebola have received lately, and the emerging threat that old pathogens may reappear as new agents of terrorism, measles virus (MV) persists as one of the leading causes of death by infectious agents worldwide, approaching the annual mortality rate of human immunodeficiency virus (HIV)-1. For most MV victims, fatality is indirect Virus-induced transient immunosuppression predisposes the individual to opportunistic infections that, left untreated, can result in mortality. In rare cases, MV may also cause progressive neurodegenerative disease. During the past five years (1998-2002), development of animal models and the application of reverse genetics and immunological assays have collectively contributed to major progress in our understanding of MV biology and pathogenesis. Nevertheless, questions and controversies remain that are the basis for future research. In this review, major advances and current debates are discussed, including MV receptor usage, the cellular basis of immunosuppression, the suspected role of MV in "nonviral" diseases such as multiple sclerosis and Paget's disease, and the controversy surrounding MV vaccine safety.

L33 ANSWER 6 OF 14 BIOTECHNO COPYRIGHT 2007 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2003:36802229 BIOTECHNO
TITLE: Elevated levels of measles antibodies in children with autism
AUTHOR: Singh V.K.; Jensen R.L.
CORPORATE SOURCE: Dr. V.K. Singh, Biotechnology Center, Utah State University, 4700 Old Main Hill, Logan, UT 84322, United States.
SOURCE: Pediatric Neurology, (2003), 28/4 (292-294), 12 reference(s).
CODEN: PNEUE2 ISSN: 0887-8994
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2003:36802229 BIOTECHNO
AB Virus-induced autoimmunity may play a causal role in autism. To examine the etiologic link of viruses in this brain disorder, we conducted a serologic study of measles virus, mumps virus, and rubella virus. Viral antibodies were measured by enzyme-linked immunosorbent assay in the serum of autistic children, normal children, and siblings of autistic children. The level of measles antibody, but not mumps or rubella antibodies, was significantly higher in autistic children as compared with normal children ($P = 0.003$) or siblings of autistic children ($P <= 0.0001$). Furthermore, immunoblotting of measles vaccine virus revealed that the antibody was directed against a protein of approximately 74 kd molecular weight. The antibody to this antigen was found in 83% of autistic children but not in normal children or siblings of autistic children. Thus autistic children have a hyperimmune response to measles virus, which in the absence of a wild type of measles infection might be a sign of an abnormal immune reaction to the vaccine strain or virus reactivation. 

L33 ANSWER 7 OF 14 BIOTECHNO COPYRIGHT 2007 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2003:37357146 BIOTECHNO
TITLE: Communicating science to the public: MMR vaccine and

autism
AUTHOR: Offit P.A.; Coffin S.E.
CORPORATE SOURCE: P.A. Offit, Division of Infectious Diseases, Children's Hospital of Philadelphia, Univ. of PA School of Medicine, 34th St. and Civic Center Blvd., Philadelphia, PA 19104, United States.
E-mail: offit@email.chop.edu
SOURCE: Vaccine, (08 DEC 2003), 22/1 (1-6), 43 reference(s)
CODEN: VACCDE ISSN: 0264-410X
DOCUMENT TYPE: Journal; General Review
COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2003:37357146 BIOTECHNO
AB Media attention and consequent public concerns about vaccine safety followed publication of a small case-series of children who developed autism after receipt of the measles-mumps-rubella (MMR) vaccine. Many well-controlled studies performed subsequently found no evidence that MMR vaccine causes autism. However, despite these studies, some parents remain concerned that the MMR vaccine is not safe. We will discuss the origins of the hypothesis that the MMR vaccine causes autism, studies performed to test the hypothesis, how these studies have been communicated to the public, and some suggested strategies for how this communication can be improved.


L33 ANSWER 8 OF 14 BIOTECHNO 
ACCESSION NUMBER: 2002:34785951 BIOTECHNO
TITLE: On the 2002 measles vaccination furore in the UK
AUTHOR: Spier R.E.
CORPORATE SOURCE: R.E. Spier, School of Biomed. and Life Sciences, University of Surrey, Guildford, Surrey GU2 7XH, United Kingdom.
E-mail: r.spier@surrey.ac.uk
SOURCE: Vaccine, (26 JUL 2002), 20/23-24 (2845-2847)
CODEN: VACCDE ISSN: 0264-410X
PUBLISHER ITEM IDENT.: S0264410X02002268
DOCUMENT TYPE: Journal; Editorial
COUNTRY: United Kingdom
LANGUAGE: English
AN 2002:34785951 BIOTECHNO

L33 ANSWER 9 OF 14 BIOTECHNO 
ACCESSION NUMBER: 2002:34492935 BIOTECHNO
TITLE: Monitoring the safety of vaccines: Assessing the risks
AUTHOR: Ellenberg S.S.; Braun M.M.
CORPORATE SOURCE: Dr. S.S. Ellenberg, Ctr. for Biologics Evaluation/Res., U.S. Food and Drug Administration, HFM-210, 1401 Rockville Pike, Rockville, MD 20852, United States.
E-mail: ellenberg@cber.FDA.gov
SOURCE: Drug Safety, (2002), 25/3 (145-152), 36 reference(s)
CODEN: DRSAEA ISSN: 0114-5916
DOCUMENT TYPE: Journal; General Review
COUNTRY: New Zealand
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2002:34492935 BIOTECHNO
AB The safety of vaccines, particularly the most widely used vaccines to which most children are exposed as infants and toddlers, has always been an extremely high priority for vaccine manufacturers and government agencies. Products intended for healthy people must be held to a high standard of safety assurance. In addition to the intense safety

assessments conducted prior to licensure, post-marketing surveillance programmes are essential to identify and study possible risks that occur too rarely to have been identified in pre-licensure studies or that occur in populations not studied in pre-licensure studies. Studying rare risks of vaccines is more complex than for therapeutic products because the exposure is virtually universal for many vaccines, ensuring occurrence simply by chance of many adverse outcomes in temporal association with vaccination. In the US the Vaccine Safety Datalink (VSD), a consortium of managed care organisations, has been established to study more rigourously possible vaccine-associated risks. These risks may be identified through reports to the Vaccine Adverse Event Reporting System (VAERS), the nationwide passive surveillance programme, as well as other sources. The combination of passive surveillance and more structured case-control or cohort studies possible in the VSD has helped to both identify new vaccine risks and to provide reassuring evidence of lack of risk in other situations where concerns have been raised.

L33 ANSWER 10 OF 14 BIOTECHNO COPYRIGHT 2007 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2002:34261208 BIOTECHNO
TITLE: Genetic and immunologic considerations in autism
AUTHOR: Korvatska E.; Van de Water J.; Anders T.F.; Gershwin M.E.
CORPORATE SOURCE: E. Korvatska, Division of Rheumatology, University of California, Davis, CA 95616, United States.
SOURCE: Neurobiology of Disease, (2002), 9/2 (107-125), 149 reference(s)
CODEN: NUDIEM ISSN: 0969-9961
DOCUMENT TYPE: Journal; General Review
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2002:34261208 BIOTECHNO
AB According to recent epidemiological surveys, autistic spectrum disorders have become recognized as common childhood psychopathologies. These life-lasting conditions demonstrate a strong genetic determinant consistent with a polygenic mode of inheritance for which several autism susceptibility regions have been identified. Parallel evidence of immune abnormalities in autistic patients argues for an implication of the immune system in pathogenesis. This review summarizes advances in the molecular genetics of autism, as well as recently emerging concerns addressing the disease incidence and triggering factors. The neurochemical and immunologic findings are analyzed in the context of a neuroimmune hypothesis for autism. Studies of disorders with established neuroimmune nature indicate multiple pathways of the pathogenesis; herein, we discuss evidence of similar phenomena in autism. .COPYRGT. 2002 Elsevier Science (USA).

L33 ANSWER 11 OF 14 BIOTECHNO COPYRIGHT 2007 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2001:32522760 BIOTECHNO
TITLE: MMR and autism: Further evidence against a causal association
AUTHOR: Farrington C.P.; Miller E.; Taylor B.
CORPORATE SOURCE: C.P. Farrington, Department of Statistics, Open University, Walton Hall, Milton Keynes MK7 6AA, United Kingdom.
E-mail: c.p.farrington@open.ac.uk
SOURCE: Vaccine, (14 JUN 2001), 19/27 (3632-3635), 8 reference(s)
CODEN: VACCDE ISSN: 0264-410X
PUBLISHER ITEM IDENT.: S0264410X01000974
DOCUMENT TYPE: Journal; Article
COUNTRY: United Kingdom

LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2001:32522760 BIOTECHNO
AB The hypothesis that MMR vaccines cause autism was first raised by reports of cases in which developmental regression occurred soon after MMR vaccination. A previous study found no evidence to support this hypothesis. It has recently been suggested that MMR vaccine might cause autism, but that the induction interval need not be short. The data from the earlier study were reanalysed to test this second hypothesis. Our results do not support this hypothesis, and provide further evidence against a causal association between MMR vaccination and autism. .COPYRGT. 2001 Elsevier Science Ltd.

L33 ANSWER 12 OF 14 BIOTECHNO COPYRIGHT 2007 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2001:34015806 BIOTECHNO
TITLE: Current overview of the pathogenesis and prophylaxis of measles with focus on practical implications
AUTHOR: Hilleman M.R.
CORPORATE SOURCE: M.R. Hilleman, Merck Institute for Vaccinology, 770 Sumneytown Pike, West Point, PA 19486, United States. E-mail: lorraine_cox@merck.com
SOURCE: Vaccine, (12 DEC 2001), 20/5-6 (651-665), 127 reference(s)
CODEN: VACCDE ISSN: 0264-410X
PUBLISHER ITEM IDENT.: S0264410X0100384X
DOCUMENT TYPE: Journal; General Review
COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2001:34015806 BIOTECHNO
AB Measles is one of the most important diseases of mankind, which is so highly contagious and evokes such persistent immunity that the virus cannot be sustained in a population of less than about 500,000 persons. The first of the licensed live virus vaccines against measles was developed empirically and was approved in 1963. It provides high level and lasting immunity and is a paradigm for solving major medical problems without really understanding them. In spite of means for control by prophylactic immunization, research on measles infection continues to be part of the effort to understand the pathogenesis of many different viruses, which may have important similarities and differences and provide important insights. Measles, usually, is spontaneously reversible and is a prime model for understanding virus-induced immunodeficiency disease (AIDS) which is rarely reversible. Much has been learned of basic immunology and vaccinology in measles through observation of the inappropriate use of vaccines of appropriate composition, and through inappropriate host response to measles vaccines of inappropriate composition. This review provides a current overview of selected highlights of measles, the virus, its immunopathogenesis, and its control by use of live virus vaccine which may lead to elimination of the disease and eventually to eradication of the virus. .COPYRGT. 2001 Elsevier Science Ltd. All rights reserved.

L33 ANSWER 13 OF 14 BIOTECHNO COPYRIGHT 2007 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2000:30186055 BIOTECHNO
TITLE: Detection and sequencing of measles virus from peripheral mononuclear cells from patients with inflammatory bowel disease and autism
AUTHOR: Kawashima H.; Mori T.; Kashiwagi Y.; Takekuma K.; Hoshika A.; Wakefield A.
CORPORATE SOURCE: Dr. H. Kawashima, Department of Paediatrics, Tokyo Medical University, 6-7-1 Nishishinjuku, Shinjuku-ku, Tokyo 160, Japan.

SOURCE: Digestive Diseases and Sciences, (2000), 45/4 (723-729), 16 reference(s)
CODEN: DDSCDJ ISSN: 0163-2116
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2000:30186055 BIOTECHNO
AB It has been reported that measles virus may be present in the intestine of patients with Crohn's disease. Additionally, a new syndrome has been reported in children with autism who exhibited developmental regression and gastrointestinal symptoms (autistic enterocolitis), in some cases soon after MMR vaccine. It is not known whether the virus, if confirmed to be present in these patients, derives from either wild strains or vaccine strains. In order to characterize the strains that may be present, we have carried out the detection of measles genomic RNA in peripheral mononuclear cells (PBMC) in eight patients with Crohn's disease, three patients with ulcerative colitis, and nine children with autistic enterocolitis. As controls, we examined healthy children and patients with SSPE, SLE, HIV-1 (a total of eight cases). RNA was purified from PBMC by Ficoll-paque, followed by reverse transcription using AMV; cDNAs were subjected to nested PCR for detection of specific regions of the hemagglutinin (H) and fusion (F) gene regions. Positive samples were sequenced directly, in nucleotides 8393-8676 (H region) or 5325- 5465 (from noncoding F to coding F region). One of eight patients with Crohn disease, one of three patients with ulcerative colitis, and three of nine children with autism, were positive. Controls were all negative. The sequences obtained from the patients with Crohn's disease shared the characteristics with wild-strain virus. The sequences obtained from the patients with ulcerative colitis and children with autism were consistent with being vaccine strains. The results were concordant with the exposure history of the patients. Persistence of measles virus was confirmed in PBMC in some patients with chronic intestinal inflammation.

L33 ANSWER 14 OF 14 BIOTECHNO 
ACCESSION NUMBER: 2000:30750728 BIOTECHNO
TITLE: Absence of chicken myelin basic protein residues in commercial formulations of MMR vaccine
AUTHOR: Afzal M.A.; Pipkin P.A.; Minor P.D.
CORPORATE SOURCE: M.A. Afzal, Division of Virology, Natl. Inst. for Biol. Std./Control, Blanche Lane, Hertfordshire EN6 3QG, United Kingdom.
E-mail: mafzal@nibsc.ac.uk
SOURCE: Vaccine, (15 OCT 2000), 19/4-5 (442-446), 13 reference(s)
CODEN: VACCDE ISSN: 0264-410X
PUBLISHER ITEM IDENT.: S0264410X00002024
DOCUMENT TYPE: Journal; Article
COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2000:30750728 BIOTECHNO
AB Several preparations of MMR vaccines and their progenitor monovalent vaccine bulks produced by two different manufacturers were examined serologically for the presence of chicken myelin basic protein (MBP) residues. The products were challenged against several commercial preparations of anti-hMBP antisera that reacted positively with the control MBP preparations of human and chicken origins. There was no evidence of the presence of MBP components in MMR vaccines or their progenitor vaccine bulks as shown by the reactivity profiles of the antibody preparations against control and test antigens. (C) 2000 Elsevier Science Ltd.

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=>  (diphtheria toxoid) and (autism or autistic)
L34             0 FILE AGRICOLA
L35             0 FILE BIOTECHNO
L36             0 FILE CONFSCI
L37             0 FILE HEALSAFE
L38             0 FILE IMSDRUGCONF
L39             0 FILE LIFESCI
L40             0 FILE PASCAL

TOTAL FOR ALL FILES
L41             0 (DIPHTHERIA TOXOID) AND (AUTISM OR AUTISTIC)

=> (pertussis or tetanus toxoid) and (autism or autistic)
L42             0 FILE AGRICOLA
L43             5 FILE BIOTECHNO
L44             0 FILE CONFSCI
L45             1 FILE HEALSAFE
L46             0 FILE IMSDRUGCONF
L47             5 FILE LIFESCI
L48             8 FILE PASCAL

TOTAL FOR ALL FILES
L49            19 (PERTUSSIS OR TETANUS TOXOID) AND (AUTISM OR AUTISTIC)

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PROCESSING COMPLETED FOR L43
L50             5 DUP REM L43 (0 DUPLICATES REMOVED)

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L50   ANSWER 1 OF 5  BIOTECHNO  COPYRIGHT 2007 Elsevier Science B.V. on STN
ACCESSION NUMBER:         2003:37329864   BIOTECHNO
TITLE:                    Public opponents of vaccination: A case study
AUTHOR:                   Leask J.; McIntyre P.
CORPORATE SOURCE:         J. Leask, Natl. Ctr. Immunisation Res./S., Children's
                          Hospital at Westmead, University of Sydney, Locked Bag
                          4001, Westmead, NSW 2145, Australia.
                          E-mail: juliel3@chw.edu.au
SOURCE:                   Vaccine, (01 DEC 2003), 21/32 (4700-4703), 28
                          reference(s)
                          CODEN: VACCDE  ISSN: 0264-410X
DOCUMENT TYPE:            Journal; Article
COUNTRY:                  United Kingdom
LANGUAGE:                 English
SUMMARY LANGUAGE:         English
AN    2003:37329864   BIOTECHNO
AB    Opposition to mass childhood vaccination is a world-wide phenomenon,
      particularly in industrialised countries. Unfounded claims about
      vaccination are perpetuated by parental lobby groups and individual
      spokespeople, some of whom have a medical or scientific background. This
      article focuses on one such spokesperson who has achieved particular
      notoriety. Dr. Viera Scheibner is a retired micropalaeontologist, without
      any formal training in health-related sciences, who tours the world
      claiming that vaccines are ineffective and dangerous and lead to a host
      of ills such as cancer and asthma. Professionals in public health or the
      clinical arena are from time to time called upon to publicly respond to
      her, or similar, claims disseminated during tours of Europe, North
      America or Australasia and in books and articles. Health professionals
      have expressed at how such spokespersons misrepresent the evidence on
      vaccine safety, resulting in the potential to undermine public confidence
      in immunisation. Media coverage, or proposed coverage, particularly of
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her more extreme claims, often makes health professionals engaged in immunisation feel obliged to respond. This paper describes Viera Scheibner's approach, which follows a repetitious path and is representative of that taken by other public opponents of immunisation. We conclude by suggesting how health professionals might respond in the public arena. .COPYRGT. 2003 Elsevier Ltd. All rights reserved.

L50 ANSWER 2 OF 5 BIOTECHNO COPYRIGHT 2007 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2003:37338229 BIOTECHNO
TITLE: The pertussis vaccine controversy in Great Britain, 1974-1986
AUTHOR: Baker J.P.
CORPORATE SOURCE: J.P. Baker, Ctr. Stud. of Med. Ethics/Hum., Duke University, Box 3040 DUMC, Durham, NC, United States.
E-mail: baker009@mc.duke.edu
SOURCE: Vaccine, (2003), 21/25-26 (4003-4010), 52 reference(s)
CODEN: VACCDE ISSN: 0264-410X
DOCUMENT TYPE: Journal; General Review
COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2003:37338229 BIOTECHNO
AB This historical essay analyzes the role played by Great Britain in the pertussis vaccine controversy of the 1970s and 1980s. Public backlash against this vaccine not only took place earlier in Britain than the United States, but also was so widespread that a series of whooping cough epidemics soon followed. As with the more recent dispute involving measles-mumps-rubella (MMR) vaccine and autism, the United Kingdom played a primary role in defining, promoting, and ultimately exporting this controversy. This essay seeks to explain this phenomenon by situating it in Britain's long history of suspicion regarding vaccines evident among both the public and the medical profession, a theme dating back to the compulsory vaccination laws of the 19th century. It argues that anti-vaccinationism, far from being simply a new development related to the public's lack of awareness of childhood vaccine-preventable illness, actually represents a revival of a much older movement.

L50 ANSWER 3 OF 5 BIOTECHNO COPYRIGHT 2007 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2003:37430627 BIOTECHNO
TITLE: Association between Thimerosal-Containing Vaccine and Autism
AUTHOR: Hviid A.; Stellfeld M.; Wohlfahrt J.; Melbye M.
CORPORATE SOURCE: A. Hviid, Danish Epidemiology Science Centre, Department of Epidemiology Research, Statens Serum Institut, Artillerivej 5, DK-2300 Copenhagen S, Denmark.
E-mail: aii@ssi.dk
SOURCE: Journal of the American Medical Association, (01 OCT 2003), 290/13 (1763-1766), 13 reference(s)
CODEN: JAMAAP ISSN: 0098-7484
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2003:37430627 BIOTECHNO
AB Context: Mercuric compounds are nephrotoxic and neurotoxic at high doses. Thimerosal, a preservative used widely in vaccine formulations, contains ethylmercury. Thus it has been suggested that childhood vaccination with thimerosal-containing vaccine could be causally related to neurodevelopmental disorders such as autism. Objective: To determine whether vaccination with a thimerosal-containing vaccine is associated with development of autism. Design, Setting, and Participants: Population-based cohort study of all children born in Denmark from January 1, 1990, until December 31, 1996 (N = 467450)

comparing children vaccinated with a thimerosal-containing vaccine with children vaccinated with a thimerosal-free formulation of the same vaccine. Main Outcome Measures: Rate ratio (RR) for autism and other autistic-spectrum disorders, including trend with dose of ethylmercury. Results During 2 986 654 person-years, we identified 440 autism cases and 787 cases of other autistic-spectrum disorders. The risk of autism and other autistic -spectrum disorders did not differ significantly between children vaccinated with thimerosal-containing vaccine and children vaccinated with thimerosal-free vaccine (RR, 0.85 [95% confidence interval {CI}, 0.60-1.20] for autism; RR, 1. 12 [95% CI, 0.88-1.43] for other autistic-spectrum disorders). Furthermore, we found no evidence of a dose-response association (increase in RR per 25 pg of ethylmercury, 0.98 [95% CI, 0.90-1.06] for autism and 1.03 [95% CI, 0.98-1.09] for other autistic-spectrum disorders). Conclusion: The results do not support a causal relationship between childhood vaccination with thimerosal-containing vaccines and development of autistic-spectrum disorders.

L50 ANSWER 4 OF 5 BIOTECHNO COPYRIGHT 2007 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2001:32234255 BIOTECHNO
TITLE: Understanding those who do not understand: A brief review of the anti-vaccine movement
AUTHOR: Poland G.A.; Jacobson R.M.
CORPORATE SOURCE: G.A. Poland, Clinical Pharmacology Unit, Department of Internal Medicine, Mayo Clinic and Foundation, 200 First Street S.W., Rochester, MN 55905, United States. E-mail: poland.gregory@mayo.edu
SOURCE: Vaccine, (21 MAR 2001), 19/17-19 (2440-2445), 25 reference(s)
CODEN: VACCDE ISSN: 0264-410X
PUBLISHER ITEM IDENT.: S0264410X00004692
DOCUMENT TYPE: Journal; Conference Article
COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2001:32234255 BIOTECHNO
AB Vaccines and the ability to prevent morbidity and mortality due to infectious diseases have been one of the greatest public health success stories. On a global level, it is one of the few cost-effective medical measures that result in universal benefit. Despite this, there is evidence of a growing anti-vaccine movement. In turn, this has, in some cases, resulted in major disruptions in vaccine programs, with resultant needless morbidity and mortality. Of interest are the factors that seem to contribute to the current trend of anti-vaccine sentiment. This paper will examine the current anti-vaccine movement and provide current examples. Finally, a review of suggestions for dealing with the anti-vaccine movement will be presented. .COPYRGT. 2001 Elsevier Science Ltd.

L50 ANSWER 5 OF 5 BIOTECHNO COPYRIGHT 2007 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2000:30446940 BIOTECHNO
TITLE: Is autism a G-alpha protein defect reversible with natural vitamin A?
AUTHOR: Megson M.N.
CORPORATE SOURCE: M.N. Megson, Medical College Virginia Hospitals, Virginia Commonwealth University, Pediatric Adolescent Ability Center, 7229 Forest Avenue, Richmond, VA 23226, United States.
SOURCE: Medical Hypotheses, (2000), 54/6 (979-983), 36 reference(s)
CODEN: MEHYDY ISSN: 0306-9877
DOCUMENT TYPE: Journal; General Review
COUNTRY: United Kingdom

```
LANGUAGE:                English
SUMMARY LANGUAGE:        English
AN     2000:30446940   BIOTECHNO
AB     Autism may be a disorder linked to the disruption of the
       G-alpha protein, affecting retinoid receptors in the brain. A study of 60
       autistic children suggests that autism may be caused by
       inserting a G-alpha protein defect, the pertussis toxin found
       in the DPT vaccine, into genetically at-risk children. This toxin
       separates the G-alpha protein from retinoid receptors. Those most at risk
       report a family history of at least one parent with a pre-existing
       G-alpha protein defect, including night blindness,
       pseudohypoparathyroidism or adenoma of the thyroid or pituitary gland.
       Natural vitamin A may reconnect the retinoid receptors critical for
       vision, sensory perception, language processing and attention.
       Autism spectrum disorders have increased from 1 in 10,000 in 1978
       to 1 in 300 in some US communities in 1999. Recent evidence indicates
       that autism is a disorder of the nervous system and the immune
       system, affecting multiple metabolic pathways. (C) 2000 Harcourt
       Publishers Ltd.


=> file .jacob
COST IN U.S. DOLLARS                          SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                               49.37      49.79

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=> (diphtheria toxoid) and (autistic or autism)
L51             0 FILE CAPLUS
L52             1 FILE BIOSIS
L53             1 FILE MEDLINE
L54             1 FILE EMBASE
L55             7 FILE USPATFULL

TOTAL FOR ALL FILES
L56            10 (DIPHTHERIA TOXOID) AND (AUTISTIC OR AUTISM)

=> d l52 ibib abs total

L52  ANSWER 1 OF 1  BIOSIS  COPYRIGHT (c) 2007 The Thomson Corporation  on STN
ACCESSION NUMBER:   2006:264741  BIOSIS
DOCUMENT NUMBER:    PREV200600264141
TITLE:              Update on vaccine liability in the United States:
                    Presentation at the National Vaccine Program Office
                    Workshop on strengthening the supply of routinely
                    recommended vaccines in the United States, 12 February
                    2002.
AUTHOR(S):          Evans, Geoffrey [Reprint Author]
CORPORATE SOURCE:   Hlth Resources and Serv Adm, Dept Hlth and Human Serv, Natl
```

Vaccine Injury Compensation Program, 5600 Fishers Ln,Rm 11C-26, Rockville, MD 20857 USA
gevans@hrsa.gov
SOURCE: Clinical Infectious Diseases, (MAR 1 2006) Vol. 42, No. Suppl. 3, pp. S130-S137,S125.
CODEN: CIDIEL. ISSN: 1058-4838.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 10 May 2006
Last Updated on STN: 10 May 2006
AB Two decades ago, a liability crisis brought on by concerns about the safety of diphtheria and tetanus toxoids and pertussis vaccine led to supply shortages and calls for rationing of the vaccine. Vaccine prices skyrocketed, and research on new products was threatened. In response, Congress created the National Vaccine Injury Compensation Program, which is tort reform legislation designed to compensate individuals quickly, easily, and generously. Since 1988, the Vaccine Injury Compensation Program has stabilized the marketplace, as evidenced by high immunization rates, stable pricing, and an increasing number of vaccine candidates in development. Although current vaccine shortages do not appear to be related to issues of liability, a new wave of tort litigation alleging that some vaccines cause autism has led to speculation that history could repeat itself.

=> dup rem
ENTER L# LIST OR (END):156
PROCESSING COMPLETED FOR L56
L57 10 DUP REM L56 (0 DUPLICATES REMOVED)

=> d l57 ibib abs total

L57 ANSWER 1 OF 10 USPATFULL on STN
ACCESSION NUMBER: 2007:11529 USPATFULL
TITLE: Biomarkers for neuropsychiatric disorders
INVENTOR(S): Bly, Michael, Ypsilanti, MI, UNITED STATES
PATENT ASSIGNEE(S): Regents of the University of Michigan, Ann Arbor, MI, UNITED STATES (U.S. corporation)

| | NUMBER | KIND | DATE | |
|---|---|---|---|---|
| PATENT INFORMATION: | US 2007009940 | A1 | 20070111 | |
| APPLICATION INFO.: | US 2006-451204 | A1 | 20060612 | (11) |

| | NUMBER | DATE |
|---|---|---|
| PRIORITY INFORMATION: | US 2005-689284P | 20050610 (60) |

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: David A. Casimir, MEDLEN & CARROLL, LLP, Suite 350, 101 Howard Street, San Francisco, CA, 94105, US
NUMBER OF CLAIMS: 6
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Page(s)
LINE COUNT: 2254
AB The present invention provides diagnostic markers of neuropsychiatric disorders (e.g., schizophrenia, schizoaffective disorder or serious mood disorders including bipolar disorder and recurrent unipolar disorder) for use in diagnosis, drug screening, therapy monitoring, research and therapeutic applications. In particular, the present invention provides SLC18A1 and TAAR2, and mutations therein, as biomarkers of neuropsychiatric disorders.

L57 ANSWER 2 OF 10 USPATFULL on STN
ACCESSION NUMBER: 2006:247166 USPATFULL
TITLE: Compositions comprising immunoreactive reagents and saponins, and methods of use thereof
INVENTOR(S): Kensil, Charlotte A, Milford, MA, UNITED STATES
PATENT ASSIGNEE(S): Antigenics, Inc., Lexington, MA, UNITED STATES, 02421 (U.S. corporation)

| | NUMBER | KIND | DATE | |
|---|---|---|---|---|
| PATENT INFORMATION: | US 2006210555 | A1 | 20060921 | |
| APPLICATION INFO.: | US 2002-499890 | A1 | 20021220 | (10) |
| | WO 2002-US40910 | | 20021220 | |
| | | | 20060408 | PCT 371 date |

| | NUMBER | DATE |
|---|---|---|
| PRIORITY INFORMATION: | US 2001-343265P | 20011221 (60) |

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: JONES DAY, 222 EAST 41ST ST, NEW YORK, NY, 10017, US
NUMBER OF CLAIMS: 56
EXEMPLARY CLAIM: 1
LINE COUNT: 1927
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to pharmaceutical compositions that are useful for the prevention and treatment of infectious diseases, primary and metastatic neoplastic diseases (i.e., cancer), neurodegenerative or amyloid diseases, or any other disease wherein the treatment of such disease would be improved by an enhanced immune response, and methods of formulating the compositions. The compositions comprise an immunoreactive reagent (i.e., an antigen binding protein comprising an antigen binding region and a region or regions of an antibody that mediate antibody dependent immunological processes) and a saponin. The present invention also relates to methods of using the compositions of the invention for the prevention and/or treatment of infectious diseases, primary and metastatic neoplastic diseases (i.e., cancer), neurodegenerative or amyloid diseases, or any other disease wherein the treatment of such disease would be improved by an enhanced immune response.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L57 ANSWER 3 OF 10 USPATFULL on STN
ACCESSION NUMBER: 2006:153717 USPATFULL
TITLE: Modified transferrin fusion proteins comprising duplicate transferrin amino or carboxy terminal domains
INVENTOR(S): Turner, Andrew J., King of Prussia, PA, UNITED STATES
Sadeghi, Homayoun, King of Prussia, PA, UNITED STATES

| | NUMBER | KIND | DATE | |
|---|---|---|---|---|
| PATENT INFORMATION: | US 2006130158 | A1 | 20060615 | |
| APPLICATION INFO.: | US 2003-515324 | A1 | 20030828 | (10) |
| | WO 2003-US26742 | | 20030828 | |
| | | | 20060119 | PCT 371 date |

| | NUMBER | DATE |
|---|---|---|
| PRIORITY INFORMATION: | US 2002-406977P | 20020830 (60) |

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MORGAN LEWIS & BOCKIUS LLP, 1111 PENNSYLVANIA AVENUE NW, WASHINGTON, DC, 20004, US

NUMBER OF CLAIMS: 41
EXEMPLARY CLAIM: 1
LINE COUNT: 13915
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Fusion proteins of transferrin and other therapeutic moieties with increased serum half-life or serum stability are disclosed. Preferred fusion proteins include those modified so that the transferrin moiety exhibits no or reduced glycosylation, binding to iron and/or binding to the transferrin receptor.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L57 ANSWER 4 OF 10 USPATFULL on STN
ACCESSION NUMBER: 2006:124654 USPATFULL
TITLE: Transferrin fusion proteins libraries
INVENTOR(S): Prior, Christopher P, King of Prussia, PA, UNITED STATES
Turner, Andrew J, King of Prussia, PA, UNITED STATES
Sadeghi, Homayoun, King of Prussia, PA, UNITED STATES

| | NUMBER | KIND | DATE | |
|---|---|---|---|---|
| PATENT INFORMATION: | US 2006105387 | A1 | 20060518 | |
| APPLICATION INFO.: | US 2003-515429 | A1 | 20030828 | (10) |
| | WO 2003-US26779 | | 20030828 | |
| | | | 20050808 | PCT 371 date |

| | NUMBER | DATE |
|---|---|---|
| PRIORITY INFORMATION: | US 2002-406977P | 20020830 (60) |
| | US 2003-485404P | 20030709 (60) |

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: COOLEY GODWARD LLP, THE BROWN BUILDING - 875 15TH STREET, NW, SUITE 800, WASHINGTON, DC, 20005-2221, US
NUMBER OF CLAIMS: 56
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 16 Drawing Page(s)
LINE COUNT: 6884
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Modified fusion proteins of transferrin and therapeutic proteins or peptides with increased serum half-life or serum stability are disclosed. Preferred fusion proteins include those modified so that the transferrin moiety exhibits no or reduced glycosylation, binding to iron and/or binding to the transferrin receptor.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L57 ANSWER 5 OF 10 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2006:264741 BIOSIS
DOCUMENT NUMBER: PREV200600264141
TITLE: Update on vaccine liability in the United States: Presentation at the National Vaccine Program Office Workshop on strengthening the supply of routinely recommended vaccines in the United States, 12 February 2002.
AUTHOR(S): Evans, Geoffrey [Reprint Author]
CORPORATE SOURCE: Hlth Resources and Serv Adm, Dept Hlth and Human Serv, Natl Vaccine Injury Compensation Program, 5600 Fishers Ln,Rm 11C-26, Rockville, MD 20857 USA
gevans@hrsa.gov
SOURCE: Clinical Infectious Diseases, (MAR 1 2006) Vol. 42, No. Suppl. 3, pp. S130-S137,S125.
CODEN: CIDIEL. ISSN: 1058-4838.

DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 10 May 2006
Last Updated on STN: 10 May 2006

AB Two decades ago, a liability crisis brought on by concerns about the safety of diphtheria and tetanus toxoids and pertussis vaccine led to supply shortages and calls for rationing of the vaccine. Vaccine prices skyrocketed, and research on new products was threatened. In response, Congress created the National Vaccine Injury Compensation Program, which is tort reform legislation designed to compensate individuals quickly, easily, and generously. Since 1988, the Vaccine Injury Compensation Program has stabilized the marketplace, as evidenced by high immunization rates, stable pricing, and an increasing number of vaccine candidates in development. Although current vaccine shortages do not appear to be related to issues of liability, a new wave of tort litigation alleging that some vaccines cause autism has led to speculation that history could repeat itself.

L57 ANSWER 6 OF 10 USPATFULL on STN
ACCESSION NUMBER: 2005:196192 USPATFULL
TITLE: Identification of etiology of autism
INVENTOR(S): Vojdani, Aristo, Los Angeles, CA, UNITED STATES

| | NUMBER | KIND | DATE |
|---|---|---|---|
| PATENT INFORMATION: | US 2005170333 | A1 | 20050804 |
| APPLICATION INFO.: | US 2004-770712 | A1 | 20040203 (10) |

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: KNOBBE MARTENS OLSON & BEAR LLP, 2040 MAIN STREET, FOURTEENTH FLOOR, IRVINE, CA, 92614, US
NUMBER OF CLAIMS: 30
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 22 Drawing Page(s)
LINE COUNT: 3959
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Disclosed herein is a method for following up a prognosis of children with autism before and after treatment with different modalities administered by their clinicians, confirming the involvement of infectious agents, dietary proteins, and toxic chemicals in development of autism. The method utilizes detection of increased amounts of antibodies against an antigen based on infectious agent, toxic chemicals, or dietary proteins. Another method utilizes detection of antibodies to a self-tissue or peptide.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L57 ANSWER 7 OF 10 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2005475531 EMBASE
TITLE: [Does vaccination cause disease?].
MACHEN IMPFUNGEN KRANK?.
AUTHOR: Zingg W.
CORPORATE SOURCE: Dr. W. Zingg, Oberarzt Medizin, Universitatskinderklinik Zurich, CH-8032 Zurich, Switzerland.
walter.zingg@kispi.unizh.ch
SOURCE: Therapeutische Umschau, (2005) Vol. 62, No. 10, pp. 665-674. .
Refs: 64
ISSN: 0040-5930 CODEN: THUMAM
COUNTRY: Switzerland
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 004 Microbiology
007 Pediatrics and Pediatric Surgery

017 Public Health, Social Medicine and Epidemiology
037 Drug Literature Index
039 Pharmacy
LANGUAGE: German
SUMMARY LANGUAGE: English; German
ENTRY DATE: Entered STN: 17 Nov 2005
Last Updated on STN: 17 Nov 2005

AB Not many inventions in medical history have influenced our society as much as vaccination. The concept is old and simple. When Edward Jenner published his work on cowpox, «variolation» was quite common. In this procedure, pus of patients with mild smallpox was transferred to healthy individuals. Meanwhile smallpox has been eradicated worldwide. Diseases such as poliomyelitis, diphtheria or tetanus almost disappeared in industrialized countries. The same happened with epiglottitis and meningitis due to Haemophilus influenzae type b (Hib) after vaccination against Hib was introduced in Switzerland in 1990. This success was possible because of routine vaccination. Immunization is a save procedure and adverse events are much lower than complications in the natural course of the prevented diseases. However vaccinations were accused to cause diseases themselves such as asthma, multiple sclerosis, diabetes mellitus, chronic arthritis or autism. Hitherto no large cohort study or case-control-study was able to proof responsibility of vaccines in any of these diseases. Public media are eager to publish early data from surveillance reports or case reports which are descriptive and never a principle of cause and effect. In large controlled trials there was no proof that vaccination causes asthma, hepatitis-B-vaccination causes multiple sclerosis or macrophagic myofasciitis, Hib-vaccination causes diabetes mellitus, rubella-vaccination causes chronic arthritis, measles-mumps-rubella-vaccination causes gait disturbance or thiomersal causes autism. These results are rarely published in newspapers or television. Thus, many caring parents are left with negative ideas about immunization. Looking for the best for their children they withhold vaccination and give way to resurgence of preventable diseases in our communities. This must be prevented. There is more evidence than expected that vaccination is safe and this can and must be told to parents. .COPYRGT. 2005 by Verlag Hans Huber.

L57 ANSWER 8 OF 10 MEDLINE on STN
ACCESSION NUMBER: 2005294112 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15795695
TITLE: A two-phased population epidemiological study of the safety of thimerosal-containing vaccines: a follow-up analysis.
AUTHOR: Geier David A; Geier Mark R
CORPORATE SOURCE: MedCon, Inc., USA.
SOURCE: Medical science monitor : international medical journal of experimental and clinical research, (2005 Apr) Vol. 11, No. 4, pp. CR160-70. Electronic Publication: 2005-03-24. Journal code: 9609063. ISSN: 1234-1010.
PUB. COUNTRY: Poland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200506
ENTRY DATE: Entered STN: 8 Jun 2005
Last Updated on STN: 29 Jun 2005
Entered Medline: 28 Jun 2005

AB BACKGROUND: Thimerosal is an ethylmercury-containing preservative in vaccines. Toxicokinetic studies have shown children received doses of mercury from thimerosal-containing vaccines (TCVs) that were in excess of safety guidelines. Previously, an ecological study showing a significant association between TCVs and neurodevelopmental disorders (NDs) in the US was published in this journal. MATERIAL/METHODS: A two phased population-based epidemiological study was undertaken. Phase one evaluated reported NDs to the Vaccine Adverse Event Reporting System

(VAERS) following thimerosal-containing Diphtheria-Tetanus-acellular-Pertussis (DTaP) vaccines in comparison to thimerosal-free DTaP vaccines administered from 1997 through 2001. Phase two evaluated the automated Vaccine Safety Datalink (VSD) for cumulative exposures to mercury from TCVs at 1-, 2-, 3-, and 6-months-of-age for infants born from 1992 through 1997 and the eventual risk of developing NDs. RESULTS: Phase one showed significantly increased risks for autism, speech disorders, mental retardation, personality disorders, and thinking abnormalities reported to VAERS following thimerosal-containing DTaP vaccines in comparison to thimerosal-free DTaP vaccines. Phase two showed significant associations between cumulative exposures to thimerosal and the following types of NDs: unspecified developmental delay, tics, attention deficit disorder (ADD), language delay, speech delay, and neurodevelopmental delays in general. CONCLUSIONS: This study showed that exposure to mercury from TCVs administered in the US was a consistent significant risk factor for the development of NDs. It is clear from these data and other recent publications linking TCVs with NDs that additional ND research should be undertaken in the context of evaluating mercury-associated exposures and thimerosal-free vaccines should be made available.

L57 ANSWER 9 OF 10 USPATFULL on STN
ACCESSION NUMBER: 2004:31195 USPATFULL
TITLE: Modified transferrin fusion proteins
INVENTOR(S): Prior, Christopher P., Philadelphia, PA, UNITED STATES
PATENT ASSIGNEE(S): BioRexis Pharmaceutical Corporation (U.S. corporation)

| | NUMBER | KIND | DATE | |
|---|---|---|---|---|
| PATENT INFORMATION: | US 2004023334 | A1 | 20040205 | |
| APPLICATION INFO.: | US 2002-231494 | A1 | 20020830 | (10) |

| | NUMBER | DATE | |
|---|---|---|---|
| PRIORITY INFORMATION: | US 2001-315745P | 20010830 | (60) |
| | US 2001-334059P | 20011130 | (60) |

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MORGAN LEWIS & BOCKIUS LLP, 1111 PENNSYLVANIA AVENUE NW, WASHINGTON, DC, 20004
NUMBER OF CLAIMS: 56
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 14 Drawing Page(s)
LINE COUNT: 15780
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Modified fusion proteins of transferrin and therapeutic proteins or peptides with increased serum half-life or serum stability are disclosed. Preferred fusion proteins include those modified so that the transferrin moiety exhibits no or reduced glycosylation, binding to iron and/or binding to the transferrin receptor.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L57 ANSWER 10 OF 10 USPATFULL on STN
ACCESSION NUMBER: 2002:66639 USPATFULL
TITLE: Compositions comprising heat shock proteins or alpha(2) macroglobulin, antigenic molecules and saponins, and methods of use thereof
INVENTOR(S): Armen, Garo H., Manhasset, NY, UNITED STATES

| | NUMBER | KIND | DATE | |
|---|---|---|---|---|
| PATENT INFORMATION: | US 2002037290 | A1 | 20020328 | |
| APPLICATION INFO.: | US 2001-909778 | A1 | 20010720 | (9) |

| | NUMBER | DATE |
|---|---|---|
| PRIORITY INFORMATION: | US 2000-223133P | 20000807 (60) |
| DOCUMENT TYPE: | Utility | |
| FILE SEGMENT: | APPLICATION | |
| LEGAL REPRESENTATIVE: | Pennie & Edmonds LLP, 1155 Avenue of the Americas, New York, NY, 10036-2711 | |
| NUMBER OF CLAIMS: | 119 | |
| EXEMPLARY CLAIM: | 1 | |
| LINE COUNT: | 4136 | |

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to pharmaceutical compositions and methods for the prevention and treatment of autoimmune diseases, infectious diseases, neurodegenerative diseases, and primary and metastatic neoplastic diseases. In the practice of the invention, the compositions are employed comprising: (a) a heat shock protein (hsp) or an alpha(2)macroglobulin (α2M); (b) a saponin; and, optionally, (c) an antigenic molecule. The antigenic molecule displays the antigenicity of an antigen of: (a) a cell that elicits an autoimmune response; (b) an agent of an infectious disease; (c) a cancerous cell; or (d) a cell or structure associated with a neurodegenerative or amyloid disease. The hsps that can be used in the practice of the invention include but are not limited to hsp70, hsp90, gp96, calreticulin, hsp 110, grp 170, and PDI, alone or in combination with each other. The antigenic molecule can be covalently or noncovalently bound to the hsp or α2M, free in solution, and/or covalently bound to the saponin. The compositions of the invention can be administered alone or in combination with the administration of antigen presenting cells sensitized with an hsp- or α2M-antigenic molecule complex.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

# EAST Search History

| Ref # | Hits | Search Query | DBs | Default Operator | Plurals | Time Stamp |
|---|---|---|---|---|---|---|
| L1 | 41 | (mercury or hg) same (autistic or autism) | US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT; IBM_TDB | OR | OFF | 2007/01/24 17:29 |
| L2 | 1 | (mercury or hg) same (autistic or autism) same (antigen or antibody or IgG or IgM or IgA) | US-PGPUB; USPAT; USOCR; FPRS; EPO; JPO; DERWENT; IBM_TDB | OR | OFF | 2007/01/24 17:30 |

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| NEWS | 5 | NOV 03 | JAPIO enhanced with IPC 8 features and functionality |
| NEWS | 6 | NOV 10 | CA/CAplus F-Term thesaurus enhanced |
| NEWS | 7 | NOV 10 | STN Express with Discover! free maintenance release Version 8.01c now available |
| NEWS | 8 | NOV 20 | CAS Registry Number crossover limit increased to 300,000 in additional databases |
| NEWS | 9 | NOV 20 | CA/CAplus to MARPAT accession number crossover limit increased to 50,000 |
| NEWS | 10 | DEC 01 | CAS REGISTRY updated with new ambiguity codes |
| NEWS | 11 | DEC 11 | CAS REGISTRY chemical nomenclature enhanced |
| NEWS | 12 | DEC 14 | WPIDS/WPINDEX/WPIX manual codes updated |
| NEWS | 13 | DEC 14 | GBFULL and FRFULL enhanced with IPC 8 features and functionality |
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| NEWS | 15 | DEC 18 | CA/CAplus patent kind codes updated |
| NEWS | 16 | DEC 18 | MARPAT to CA/CAplus accession number crossover limit increased to 50,000 |
| NEWS | 17 | DEC 18 | MEDLINE updated in preparation for 2007 reload |
| NEWS | 18 | DEC 27 | CA/CAplus enhanced with more pre-1907 records |
| NEWS | 19 | JAN 08 | CHEMLIST enhanced with New Zealand Inventory of Chemicals |
| NEWS | 20 | JAN 16 | CA/CAplus Company Name Thesaurus enhanced and reloaded |
| NEWS | 21 | JAN 16 | IPC version 2007.01 thesaurus available on STN |
| NEWS | 22 | JAN 16 | WPIDS/WPINDEX/WPIX enhanced with IPC 8 reclassification data |
| NEWS | 23 | JAN 22 | CA/CAplus updated with revised CAS roles |
| NEWS | 24 | JAN 22 | CA/CAplus enhanced with patent applications from India |

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| NEWS IPC8 | For general information regarding STN implementation of IPC 8 |
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L1 8 FILE CAPLUS
L2 5 FILE BIOSIS
L3 10 FILE MEDLINE
L4 17 FILE EMBASE
L5 73 FILE USPATFULL

TOTAL FOR ALL FILES
L6 113 (AUTISM OR AUTISTIC) AND (MERCURY OR MERCURIAL) AND (ANTIBODY OR IGG OR IGA OR IGM OR ANTIGEN OR PROTEIN)

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=> l6 and diagnos4
L7 5506253 FILE CAPLUS
L8 2202698 FILE BIOSIS
L9 2385266 FILE MEDLINE
L10 1469957 FILE EMBASE
L11 4628160 FILE USPATFULL

TOTAL FOR ALL FILES
L12 16192334 4

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PROCESSING COMPLETED FOR L2
PROCESSING COMPLETED FOR L3
PROCESSING COMPLETED FOR L4
L13 23 DUP REM L1-L4 (17 DUPLICATES REMOVED)

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L13 ANSWER 1 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 2006:1086506 CAPLUS
DOCUMENT NUMBER: 145:370901
TITLE: Metallothioneins: mercury species-specific induction and their potential role in attenuating neurotoxicity
AUTHOR(S): Aschner, Michael; Syversen, Tore; Souza, Diogo O.; Rocha, Joao B. T.
CORPORATE SOURCE: Departments of Pediatrics, Pharmacology, Vanderbilt University Medical Center, Nashville, TN, USA
SOURCE: Experimental Biology and Medicine (Maywood, NJ, United States) (2006), 231(9), 1468-1473
CODEN: EBMMBE; ISSN: 1535-3702
PUBLISHER: Society for Experimental Biology and Medicine
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. Metallothionein (MT) proteins are widespread in bacteria, fungi, plants, and eukaryotic species. They are of low mol. weight (6-7 kDa) and of the 60+ amino acid residues, 20 are cysteines. Functions attributed to MTs include the sequestration and dispersal of metal ions, primarily in Zn and Cu homeostasis; regulation of the biosynthesis and activity of Zn metalloproteins, most notably Zn-dependent transcription factors; and cellular cytoprotection from reactive oxygen species, ionizing radiation, electrophilic anticancer drugs and mutagens, and metals. Observations on the abundance of MTs within the central nervous system (CNS) and the identification of a brain-specific isoform, MT-III, suggest that it might have important neurophysiol. and neuromodulatory functions. Reinforced by the potential involvement of MT-III in a number of neurodegenerative disorders, the role of MTs in the CNS has become an intense focus of scientific pursuit. This manuscript represents a survey on the ability of MTs to modulate Hg neurotoxicity, a neurotoxin that was implied to play an etiol. role in Minamata disease, erethism, and autism, just to name a few.
REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 2 OF 23 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2006496402 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16870260
TITLE: Cultured lymphocytes from autistic children and non-autistic siblings up-regulate heat shock protein RNA in response to thimerosal challenge.
AUTHOR: Walker Stephen J; Segal Jeffrey; Aschner Michael
CORPORATE SOURCE: Department of Physiology and Pharmacology, Wake Forest University School of Medicine, Winston-Salem, NC 27156, USA.. swalker@wfubmc.edu
SOURCE: Neurotoxicology, (2006 Sep) Vol. 27, No. 5, pp. 685-92.
Electronic Publication: 2006-06-16.
Journal code: 7905589. ISSN: 0161-813X.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200610
ENTRY DATE: Entered STN: 22 Aug 2006
Last Updated on STN: 1 Nov 2006
Entered Medline: 31 Oct 2006
AB There are reports suggesting that some autistic children are unable to mount an adequate response following exposure to environmental toxins. This potential deficit, coupled with the similarity in clinical presentations of autism and some heavy metal toxicities, has led to the suggestion that heavy metal poisoning might play a role in the

etiology of autism in uniquely susceptible individuals. Thimerosal, an anti-microbial preservative previously added routinely to childhood multi-dose vaccines, is composed of 49.6% ethyl mercury . Based on the levels of this toxin that children receive through routine immunization schedules in the first years of life, it has been postulated that thimerosal may be a potential triggering mechanism contributing to autism in susceptible individuals. One potential risk factor in these individuals may be, an inability to adequately up-regulate metallothionein (MT) biosynthesis in response to presentation of a heavy metal challenge. To investigate this hypothesis, cultured lymphocytes (obtained from the Autism Genetic Resource Exchange, AGRE) from autistic children and non-autistic siblings were challenged with either 10 microM ethyl mercury, 150 microM zinc, or fresh media (control). Following the challenge, total RNA was extracted and used to query "whole genome" DNA microarrays. Cultured lymphocytes challenged with zinc responded with an impressive up-regulation of MT transcripts (at least nine different MTs were over-expressed) while cells challenged with thimerosal responded by up-regulating numerous heat shock protein transcripts, but not MTs. Although there were no apparent differences between autistic and non-autistic sibling responses in this very small sampling group, the differences in expression profiles between those cells treated with zinc versus thimerosal were dramatic. Determining cellular response, at the level of gene expression, has important implications for the understanding and treatment of conditions that result from exposure to neurotoxic compounds.

L13 ANSWER 3 OF 23 EMBASE 

ACCESSION NUMBER: 2006419048 EMBASE
TITLE: Will widespread human papillomavirus prophylactic vaccination change sexual practices of adolescent and young adult women in America?.
AUTHOR: Monk B.J.; Wiley D.J.
CORPORATE SOURCE: Dr. B.J. Monk, Division of Gynecologic Oncology, University of California, Irvine, Chao Family Comprehensive Cancer Center, 101 The City Drive, Orange, CA 92868, United States. bjmonk@uci.edu
SOURCE: Obstetrics and Gynecology, (2006) Vol. 108, No. 2, pp. 420-424. .
Refs: 31
ISSN: 0029-7844 CODEN: OBGNAS
PUBLISHER IDENT.: 000062502006080000026
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 010 Obstetrics and Gynecology
017 Public Health, Social Medicine and Epidemiology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 8 Sep 2006
Last Updated on STN: 8 Sep 2006

AB Two virus-like particle human papillomavirus (HPV) vaccines have been shown to be nearly 100% effective in preventing type-specific persistent HPV infections and associated type-specific high-grade cervical intraepithelial neoplasia (CIN). Recently, it has been hypothesized that the administration of this vaccine to young girls in the United States might increase sexual promiscuity among adolescent women and/or young adults. Thus, it has been suggested that focused vaccine strategies either based on the risk of CIN or gender might be more rational or cost-effective. However, such strategies are unlikely to completely eradicate the burden of this disease and decrease the cost of cervical

cancer screening. The suggestion that widespread vaccination will alter sexual practices is refuted and the rationale for the vaccination of all girls and boys is outlined. .COPYRGT. 2006 by The American College of Obstetricians and Gynecologists. Published by Lippincott Williams & Wilkins.

L13 ANSWER 4 OF 23 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2006358969 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16771783
TITLE: Assessment of metallothionein and antibodies to metallothionein in normal and autistic children having exposure to vaccine-derived thimerosal.
AUTHOR: Singh Vijendra K; Hanson Jeff
CORPORATE SOURCE: Department of Biology, Utah State University, Logan, UT 84322, USA.. singhvk@cc.usu.edu
SOURCE: Pediatric allergy and immunology : official publication of the European Society of Pediatric Allergy and Immunology, (2006 Jun) Vol. 17, No. 4, pp. 291-6.
Journal code: 9106718. ISSN: 0905-6157.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200608
ENTRY DATE: Entered STN: 15 Jun 2006
Last Updated on STN: 10 Aug 2006
Entered Medline: 9 Aug 2006

AB Allergic autoimmune reaction after exposure to heavy metals such as mercury may play a causal role in autism, a developmental disorder of the central nervous system. As metallothionein (MT) is the primary metal-detoxifying protein in the body, we conducted a study of the MT protein and antibodies to metallothionein (anti-MT) in normal and autistic children whose exposure to mercury was only from thimerosal-containing vaccines. Laboratory analysis by immunoassays revealed that the serum level of MT did not significantly differ between normal and autistic children. Furthermore, autistic children harboured normal levels of anti-MT, including antibodies to isoform MT-I (anti-MT-I) and MT-II (anti-MT-II), without any significant difference between normal and autistic children. Our findings indicate that because autistic children have a normal profile of MT and anti-MT, the mercury-induced autoimmunity to MT may not be implicated in the pathogenesis of autism.

L13 ANSWER 5 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2006323177 EMBASE
TITLE: Oxidative stress in autism.
AUTHOR: Chauhan A.; Chauhan V.
CORPORATE SOURCE: A. Chauhan, NYS Institute for Basic Research in Developmental Disabilities, 1050 Forest Hill Road, Staten Island, NY 10314, United States. Abhachauhan@aol.com
SOURCE: Pathophysiology, (2006) Vol. 13, No. 3, pp. 171-181. .
Refs: 146
ISSN: 0928-4680 CODEN: PTHOE7
PUBLISHER IDENT.: S 0928-4680(06)00053-8
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
007 Pediatrics and Pediatric Surgery
008 Neurology and Neurosurgery
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English

SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 1 Aug 2006
Last Updated on STN: 1 Aug 2006

AB Autism is a severe developmental disorder with poorly understood etiology. Oxidative stress in autism has been studied at the membrane level and also by measuring products of lipid peroxidation, detoxifying agents (such as glutathione), and antioxidants involved in the defense system against reactive oxygen species (ROS). Lipid peroxidation markers are elevated in autism, indicating that oxidative stress is increased in this disease. Levels of major antioxidant serum proteins, namely transferrin (iron-binding protein) and ceruloplasmin (copper-binding protein), are decreased in children with autism. There is a positive correlation between reduced levels of these proteins and loss of previously acquired language skills in children with autism. The alterations in ceruloplasmin and transferrin levels may lead to abnormal iron and copper metabolism in autism. The membrane phospholipids, the prime target of ROS, are also altered in autism. The levels of phosphatidylethanolamine (PE) are decreased, and phosphatidylserine (PS) levels are increased in the erythrocyte membrane of children with autism as compared to their unaffected siblings. Several studies have suggested alterations in the activities of antioxidant enzymes such as superoxide dismutase, glutathione peroxidase, and catalase in autism. Additionally, altered glutathione levels and homocysteine/methionine metabolism, increased inflammation, excitotoxicity, as well as mitochondrial and immune dysfunction have been suggested in autism. Furthermore, environmental and genetic factors may increase vulnerability to oxidative stress in autism . Taken together, these studies suggest increased oxidative stress in autism that may contribute to the development of this disease. A mechanism linking oxidative stress with membrane lipid abnormalities, inflammation, aberrant immune response, impaired energy metabolism and excitotoxicity, leading to clinical symptoms and pathogenesis of autism is proposed. 

L13 ANSWER 6 OF 23 CAPLUS 
ACCESSION NUMBER: 2005:698214 CAPLUS
DOCUMENT NUMBER: 143:171341
TITLE: Methods for detecting infections, toxic chemicals and dietary peptides binding to lymphocyte receptors and tissue enzymes as instigators of autoimmunity in autism
INVENTOR(S): Vojdani, Aristo
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 89 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| US 2005170333 | A1 | 20050804 | US 2004-770712 | 20040203 |
| PRIORITY APPLN. INFO.: | | | US 2004-770712 | 20040203 |

AB The present invention provides methods for diagnosis and following up a prognosis of children with autism before and after treatment with different modalities administered by their clinicians, confirming the involvement of infectious agents, dietary proteins, and toxic chems. in development of autism. In particular, methods for detecting infections, toxic chems. and dietary peptides binding to lymphocyte receptors and tissue enzymes as instigators of autoimmunity in autism are described. The method utilizes detection of increased

amts. of antibodies against an antigen based on infectious agent, toxic chems., or dietary proteins. Another method utilizes detection of antibodies to a self-tissue or peptide.

L13 ANSWER 7 OF 23 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2006:265087 BIOSIS
DOCUMENT NUMBER: PREV200600265226
TITLE: Immunological findings in autism.
AUTHOR(S): Cohly, Hari Har Parshad [Reprint Author]; Panja, Asit
CORPORATE SOURCE: Jackson State Univ, Dept Biol, Jackson, MS 39217 USA
SOURCE: Dhossche, DM [Editor]. Int. Rev. Neurobiol., (2005) pp. 317-341. International Review of Neurobiology. Publisher: ELSEVIER ACADEMIC PRESS INC, 525 B STREET, SUITE 1900, SAN DIEGO, CA 92101-4495 USA. Series: INTERNATIONAL REVIEW OF NEUROBIOLOGY. CODEN: IRNEAE. ISSN: 0074-7742. ISBN: 0-12-366872-7(H).
DOCUMENT TYPE: Book; (Book Chapter)
LANGUAGE: English
ENTRY DATE: Entered STN: 10 May 2006
Last Updated on STN: 10 May 2006

AB Autism is a disorder of neurobiological origin characterized by impairment of contact and communications. Typical symptoms of autism include extreme withdrawal and in abnormal absorption in fantasy, accompanied by delusion, hallucination, and an inability to communicate verbally or to otherwise relate to people. The cause of autism remains unknown. However, there are several factors including infectious, neurological, metabolic, environmental, and immunologic origin that have been thought to be involved in the disease development process of autism. The cellular entities playing a role in the pathologic processes in the autistic brain are the neurons, glial cells, endothelial cells, microglial cells, and astrocytes with blood brain barrier permeability playing all important role for the trafficking of the immune cells and mediators. In this chapter immunologic findings on autism are discussed. Particular emphasis is made on the aspects of immunological dysfunctions and inflammation as the two important immunological principles contributing to the diseases process in autism.

L13 ANSWER 8 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2005485455 EMBASE
TITLE: Preservatives in DPT vaccine.
AUTHOR: Paul Y.; Parthasarathy A.
CORPORATE SOURCE: Y. Paul, A-D-7, Devi Magr, Bani Park, Jaipur 302 016, India. eryashpaul2003@yahoo.com
SOURCE: Indian Pediatrics, (2005) Vol. 42, No. 10, pp. 1006-1007. ISSN: 0019-6061 CODEN: INPDAR
COUNTRY: India
DOCUMENT TYPE: Journal; Note
FILE SEGMENT: 007 Pediatrics and Pediatric Surgery
026 Immunology, Serology and Transplantation
037 Drug Literature Index
038 Adverse Reactions Titles
039 Pharmacy
LANGUAGE: English
ENTRY DATE: Entered STN: 17 Nov 2005
Last Updated on STN: 17 Nov 2005
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L13 ANSWER 9 OF 23 MEDLINE on STN
ACCESSION NUMBER: 2006125519 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16512356
TITLE: Immunological findings in autism.

AUTHOR: Cohly Hari Har Parshad; Panja Asit
CORPORATE SOURCE: Department of Biology, Jackson State University, Mississippi 39217, USA.
SOURCE: International review of neurobiology, (2005) Vol. 71, pp. 317-41. Ref: 135
Journal code: 0374740. ISSN: 0074-7742.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200603
ENTRY DATE: Entered STN: 4 Mar 2006
Last Updated on STN: 30 Mar 2006
Entered Medline: 29 Mar 2006

AB The immunopathogenesis of autism is presented schematically in Fig. 1. Two main immune dysfunctions in autism are immune regulation involving pro-inflammatory cytokines and autoimmunity. Mercury and an infectious agent like the measles virus are currently two main candidate environmental triggers for immune dysfunction in autism. Genetically immune dysfunction in autism involves the MHC region, as this is an immunologic gene cluster whose gene products are Class I, II, and III molecules. Class I and II molecules are associated with antigen presentation. The antigen in virus infection initiated by the virus particle itself while the cytokine production and inflammatory mediators are due to the response to the putative antigen in question. The cell-mediated immunity is impaired as evidenced by low numbers of CD4 cells and a concomitant T-cell polarity with an imbalance of Th1/Th2 subsets toward Th2. Impaired humoral immunity on the other hand is evidenced by decreased IgA causing poor gut protection. Studies showing elevated brain specific antibodies in autism support an autoimmune mechanism. Viruses may initiate the process but the subsequent activation of cytokines is the damaging factor associated with autism. Virus specific antibodies associated with measles virus have been demonstrated in autistic subjects. Environmental exposure to mercury is believed to harm human health possibly through modulation of immune homeostasis. A mercury link with the immune system has been postulated due to the involvement of postnatal exposure to thimerosal, a preservative added in the MMR vaccines. The occupational hazard exposure to mercury causes edema in astrocytes and, at the molecular level, the CD95/Fas apoptotic signaling pathway is disrupted by Hg2+. Inflammatory mediators in autism usually involve activation of astrocytes and microglial cells. Proinflammatory chemokines (MCP-1 and TARC), and an anti-inflammatory and modulatory cytokine, TGF-beta1, are consistently elevated in autistic brains. In measles virus infection, it has been postulated that there is immune suppression by inhibiting T-cell proliferation and maturation and downregulation MHC class II expression. Cytokine alteration of TNF-alpha is increased in autistic populations. Toll-like-receptors are also involved in autistic development. High NO levels are associated with autism. Maternal antibodies may trigger autism as a mechanism of autoimmunity. MMR vaccination may increase risk for autism via an autoimmune mechanism in autism. MMR antibodies are significantly higher in autistic children as compared to normal children, supporting a role of MMR in autism. Autoantibodies (IgG isotype) to neuron-axon filament protein (NAFP) and glial fibrillary acidic protein (GFAP) are significantly increased in autistic patients (Singh et al., 1997). Increase in Th2 may explain the increased autoimmunity, such as the findings of antibodies to MBP and neuronal axonal filaments in the brain. There is further evidence that there are other participants in the autoimmune phenomenon. (Kozlovskaia et al., 2000).

The possibility of its involvement in autism cannot be ruled out. Further investigations at immunological, cellular, molecular, and genetic levels will allow researchers to continue to unravel the immunopathogenic mechanisms' associated with autistic processes in the developing brain. This may open up new avenues for prevention and/or cure of this devastating neurodevelopmental disorder.

L13 ANSWER 10 OF 23 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 2005155328 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15789284
TITLE: [Amalgam risk assessment with coverage of references up to 2005].
Amalgam: Eine Risikobewertung unter Berucksichtigung der neuen Literatur bis 2005.
AUTHOR: Mutter J; Naumann J; Walach H; Daschner F
CORPORATE SOURCE: Institut fur Umweltmedizin und Krankenhaushygiene, Universitatsklinik Freiburg.. joachim.mutter@uniklinik-freiburg.de
SOURCE: Gesundheitswesen (Bundesverband der rzte des ffentlichen Gesundheitsdienstes (Germany)), (2005 Mar) Vol. 67, No. 3, pp. 204-16. Ref: 248
Journal code: 9204210. ISSN: 0941-3790.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: German
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200505
ENTRY DATE: Entered STN: 25 Mar 2005
Last Updated on STN: 27 May 2005
Entered Medline: 26 May 2005

AB Amalgam, which has been in use in dentistry for 150 years, consists of 50 % elemental mercury and a mixture of silver, tin, copper and zinc. Minute amounts of mercury vapour are released continuously from amalgam. Amalgam contributes substantially to human mercury load. Mercury accumulates in some organs, particularly in the brain, where it can bind to protein more tightly than other heavy metals (e. g. lead, cadmium). Therefore, the elimination half time is assumed to be up to 1 - 18 years in the brain and bones. Mercury is assumed to be one of the most toxic non-radioactive elements. There are pointers to show that mercury vapour is more neurotoxic than methyl-mercury in fish. Review of recent literature suggests that mercury from dental amalgam may lead to nephrotoxicity, neurobehavioural changes, autoimmunity, oxidative stress, autism, skin and mucosa alterations or non-specific symptoms and complaints. The development of Alzheimer's disease or multiple sclerosis has also been linked to low-dose mercury exposure. There may be individual genetical or acquired susceptibilities for negative effects from dental amalgam. Mercury levels in the blood, urine or other biomarkers do not reflect the mercury load in critical organs. Some studies regarding dental amalgam reveal substantial methodical flaws. Removal of dental amalgam leads to permanent improvement of various chronic complaints in a relevant number of patients in various trials. Summing up, available data suggests that dental amalgam is an unsuitable material for medical, occupational and ecological reasons.

L13 ANSWER 11 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:452933 CAPLUS
DOCUMENT NUMBER: 141:37230
TITLE: Nuclear receptors as diagnostic and risk markers for disease and as targets for therapy
INVENTOR(S): Gaitanaris, George A.; Bergmann, John E.; Gracerov, Alexander; Hohmann, John; Li, Fusheng; Madisen, Linda;

Mcilwain, Kellie L.; Pavlova, Maria N.; Vassilatis, Demetri; Zeng, Hongkui

PATENT ASSIGNEE(S): Nura, Inc., USA

SOURCE: PCT Int. Appl., 508 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| WO 2004045369 | A2 | 20040603 | WO 2003-US36229 | 20031112 |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW | | | | |
| RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| AU 2003295500 | A1 | 20040615 | AU 2003-295500 | 20031112 |

PRIORITY APPLN. INFO.: US 2002-426305P P 20021114
WO 2003-US36229 W 20031112

AB Methods of using nuclear receptors as diagnostic markers for disease and for increased risk of disease and in the development of therapeutics for treatment of such diseases are described. The proteins and the genes encoding them may be used in diagnosis. Transgenic animals carrying the human genes for these receptors may be used in screening for effectors. The invention also provides methods for identifying compds. (e.g., agonists or antagonists) using the nuclear receptor polypeptides and polynucleotides of the invention, and for treating conditions associated with nuclear receptor dysfunction with the nuclear receptor polypeptides, polynucleotides, or identified compds. The invention also provides diagnostic assays for detecting diseases or disorders associated with inappropriate nuclear receptor activity or levels.

L13 ANSWER 12 OF 23 EMBASE 

ACCESSION NUMBER: 2004147991 EMBASE

TITLE: Childhood Immunizations and Chronic Illness.

AUTHOR: Levitsky L.L.

CORPORATE SOURCE: Dr. L.L. Levitsky, Pediatric Endocrine Unit, Massachusetts General Hospital, Harvard Medical School, Boston, MA, United States

SOURCE: New England Journal of Medicine, (1 Apr 2004) Vol. 350, No. 14, pp. 1380-1382.
Refs: 6
ISSN: 0028-4793 CODEN: NEJMAG

COUNTRY: United States

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 004 Microbiology
007 Pediatrics and Pediatric Surgery
017 Public Health, Social Medicine and Epidemiology
037 Drug Literature Index
038 Adverse Reactions Titles

LANGUAGE: English

ENTRY DATE: Entered STN: 22 Apr 2004
Last Updated on STN: 22 Apr 2004

DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L13 ANSWER 13 OF 23 EMBASE 

reserved on STN
ACCESSION NUMBER: 2004186624 EMBASE
TITLE: Thimerosal and autism? A plausible hypothesis that should not be dismissed.
AUTHOR: Blaxill M.F.; Redwood L.; Bernard S.
CORPORATE SOURCE: M.F. Blaxill, 22 Fayerweather Street, Cambridge, MA 02138, United States. blaxill@comcast.net
SOURCE: Medical Hypotheses, (2004) Vol. 62, No. 5, pp. 788-794. .
Refs: 52
ISSN: 0306-9877 CODEN: MEHYDY
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 008 Neurology and Neurosurgery
037 Drug Literature Index
052 Toxicology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 28 May 2004
Last Updated on STN: 28 May 2004

AB The autism-mercury hypothesis first described by Bernard et al. has generated much interest and controversy. The Institute of Medicine (IOM) reviewed the connection between mercury -containing vaccines and neurodevelopmental disorders, including autism. They concluded that the hypothesis was biologically plausible but that there was insufficient evidence to accept or reject a causal connection and recommended a comprehensive research program. Without citing new experimental evidence, a number of observers have offered opinions on the subject, some of which reject the IOM's conclusions. In a recent review, Nelson and Bauman argue that a link between the preservative thimerosal, the source of the mercury in childhood vaccines, is improbable. In their defense of thimerosal, these authors take a narrow view of the original hypothesis, provide no new evidence, and rely on selective citations and flawed reasoning. We provide evidence here to refute the Nelson and Bauman critique and to defend the autism-mercury hypothesis. .COPYRGT. 2004 Elsevier Ltd. All rights reserved.

L13 ANSWER 14 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 5
ACCESSION NUMBER: 2004:674310 CAPLUS
DOCUMENT NUMBER: 142:22062
TITLE: Detection of Antinuclear and Antilaminin Antibodies in Autistic Children Who Received Thimerosal-Containing Vaccines
AUTHOR(S): Singh, Vijendra K.; Rivas, Wyatt H.
CORPORATE SOURCE: Department of Biology, Utah State University, Logan, UT, USA
SOURCE: Journal of Biomedical Science (Basel, Switzerland) (2004), 11(5), 607-610
CODEN: JBCIEA; ISSN: 1021-7770
PUBLISHER: S. Karger AG
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Autism, a neurodevelopmental disorder, may involve autoimmune pathogenesis. Since mercury is potentially a risk factor for autoimmunity, we conducted a study of mercury-induced antinuclear and antilaminin antibodies in autistic and normal children who had been pre-administered with thimerosal-containing vaccines. Laboratory anal. by different immunoassays showed that the serum level of these two autoimmune markers did not significantly differ between autistic and normal children. This finding suggests that the mercury as in thimerosal-containing vaccines is likely not related to autoimmune phenomenon in autism.

REFERENCE COUNT: 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 15 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 6
ACCESSION NUMBER: 2003:649270 CAPLUS
DOCUMENT NUMBER: 140:89124
TITLE: Reduced Levels of Mercury in First Baby Haircuts of Autistic Children
AUTHOR(S): Holmes, Amy S.; Blaxill, Mark F.; Haley, Boyd E.
CORPORATE SOURCE: Baton Rouge, LA, USA
SOURCE: International Journal of Toxicology (2003), 22(4), 277-285
CODEN: IJTOFN; ISSN: 1091-5818
PUBLISHER: Taylor & Francis, Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Reported rates of autism have increased sharply in the United States and the United Kingdom. One possible factor underlying these increases is increased exposure to mercury through thimerosal-containing vaccines, but vaccine exposures need to be evaluated in the context of cumulative exposures during gestation and early infancy. Differential rates of postnatal mercury elimination may explain why similar gestational and infant exposures produce variable neurol. effects. First baby haircut samples were obtained from 94 children diagnosed with autism using Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM IV) criteria and 45 age- and gender-matched controls. Information on diet, dental amalgam fillings, vaccine history, Rho D Ig administration, and autism symptom severity was collected through a maternal survey questionnaire and clin. observation. Hair mercury levels in the autistic group were 0.47 ppm vs. 3.63 ppm in controls, a significant difference. The mothers in the autistic group had significantly higher levels of mercury exposure through Rho D Ig injections and amalgam fillings than control mothers. Within the autistic group, hair mercury levels varied significantly across mildly, moderately, and severely autistic children, with mean group levels of 0.79, 0.46, and 0.21 ppm, resp. Hair mercury levels among controls were significantly correlated with the number of the mothers' amalgam fillings and their fish consumption as well as exposure to mercury through childhood vaccines, correlations that were absent in the autistic group. Hair excretion patterns among autistic infants were significantly reduced relative to control. These data cast doubt on the efficacy of traditional hair anal. as a measure of total mercury exposure in a subset of the population. In light of the biol. plausibility of mercury's role in neurodevelopmental disorders, the present study provides further insight into one possible mechanism by which early mercury exposures could increase the risk of autism.

REFERENCE COUNT: 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 16 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 7
ACCESSION NUMBER: 2004:44082 CAPLUS
DOCUMENT NUMBER: 140:216004
TITLE: Infections, toxic chemicals and dietary peptides binding to lymphocyte receptors and tissue enzymes are major instigators of autoimmunity in autism
AUTHOR(S): Vojdani, A.; Pangborn, J. B.; Vojdani, E.; Cooper, E. L.
CORPORATE SOURCE: Laboratory of Comparative Neuroimmunology, Department of Neurobiology, David Geffen School of Medicine at UCLA, University of California, Los Angeles, Los Angeles, CA, 90095, USA
SOURCE: International Journal of Immunopathology and Pharmacology (2003), 16(3), 189-199
CODEN: IJIPE4; ISSN: 0394-6320

PUBLISHER: Biolife s.a.s.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Similar to many complex autoimmune diseases, genetic and environmental factors including diet, infection and xenobiotics play a critical role in the development of autism. In this study, we postulated that infectious agent antigens such as streptokinase, dietary peptides (gliadin and casein) and Et mercury (xenobiotic) bind to different lymphocyte receptors and tissue enzyme (DPP IV or CD26). We assessed this hypothesis first by measuring IgG, IgM and IgA antibodies against CD26, CD69, streptokinase (SK), gliadin and casein peptides and against Et mercury bound to human serum albumin in patients with autism. A significant percentage of children with autism developed anti-SK, anti-gliadin and casein peptides and anti-Et mercury antibodies, concomitant with the appearance of anti-CD26 and anti-CD69 autoantibodies. These antibodies are synthesized as a result of SK, gliadin, casein and Et mercury binding to CD26 and CD69, indicating that they are specific. Immune absorption demonstrated that only specific antigens, like CD26, were capable of significantly reducing serum anti-CD26 levels. However, for direct demonstration of SK, gliadin, casein and Et mercury to CD26 or CD69, microtiter wells were coated with CD26 or CD69 alone or in combination with SK, gliadin, casein or Et mercury and then reacted with enzyme labeled rabbit anti-CD26 or anti-CD69. Adding these mols. to CD26 or CD69 resulted in 28-86% inhibition of CD26 or CD69 binding to anti-CD26 or anti-CD69 antibodies. The highest % binding of these antigens or peptides to CD26 or CD69 was attributed to SK and the lowest to casein peptides. We, therefore, propose that bacterial antigens (SK), dietary peptides (gliadin, casein) and Thimerosat (Et mercury) in individuals with pre-disposing HLA mols.; bind to CD26 or CD69 and induce antibodies against these mols. In conclusion, this study is apparently the first to demonstrate that dietary peptides, bacterial toxins and xenobiotics bind to lymphocyte receptors and/or tissue enzymes, resulting in autoimmune reaction in children with autism.
REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 17 OF 23 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2003:438158 BIOSIS
DOCUMENT NUMBER: PREV200300438158
TITLE: Structural difference of MTF-1 in some autistic individuals.
AUTHOR(S): Russo, A. J. [Reprint Author]; Gilbride, Rebecca [Reprint Author]
CORPORATE SOURCE: Department of Science, Mount Saint Mary's College, Emmitsburg, MD, 21727, USA
SOURCE: Journal of the Pennsylvania Academy of Science, (May 2003) Vol. 77, No. 1, pp. 3-6. print.
CODEN: JPSCEY. ISSN: 1044-6753.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Sep 2003
Last Updated on STN: 24 Sep 2003
AB Autism is a type of Pervasive Developmental Disorder (PDD) that affects one in five hundred Americans. Metallothionein (MT), a low molecular weight cysteine rich protein has been shown to have a protective effect against heavy metal toxicity such as mercury, zinc and cadmium. Transcription of mammalian metallothionein genes is regulated by metal-regulatory transcription factor-1 (MTF-1), which binds to the cis-acting regulatory sequences of the MT promotor, termed metal-responsive elements (MRE's). In a preliminary study, we compared a

239 bp fragment of human MTF-I in autistic and non-autistic individuals. PCR with MTF-1 primers and DNA from non-autistic individuals resulted in the expected product of 239 bp. Only two of seven DNA samples from autistic individuals yielded the expected product ($p<0.05$).

L13 ANSWER 18 OF 23 MEDLINE on STN DUPLICATE 8
ACCESSION NUMBER: 2002732930 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12495373
TITLE: Autism, an extreme challenge to integrative medicine. Part 2: medical management.
AUTHOR: Kidd Parris M
SOURCE: Alternative medicine review : a journal of clinical therapeutic, (2002 Dec) Vol. 7, No. 6, pp. 472-99. Ref: 130
Journal code: 9705340. ISSN: 1089-5159.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Consumer Health
ENTRY MONTH: 200303
ENTRY DATE: Entered STN: 27 Dec 2002
Last Updated on STN: 7 Mar 2003
Entered Medline: 6 Mar 2003

AB Autism and allied autistic spectrum disorders (ASD) present myriad behavioral, clinical, and biochemical abnormalities. Parental participation, advanced testing protocols, and eclectic treatment strategies have driven progress toward cure. Behavioral modification and structured education are beneficial but insufficient. Dietary restrictions, including removal of milk and other casein dairy products, wheat and other gluten sources, sugar, chocolate, preservatives, and food coloring are beneficial and prerequisite to benefit from other interventions. Individualized IgG or IgE testing can identify other troublesome foods but not non-immune mediated food sensitivities. Gastrointestinal improvement rests on controlling Candida and other parasites, and using probiotic bacteria and nutrients to correct dysbiosis and decrease gut permeability. Detoxification of mercury and other heavy metals by DMSA/DMPS chelation can have marked benefit. Documented sulfoxidation-sulfation inadequacies call for sulfur-sulfhydryl repletion and other liver p450 support. Many nutrient supplements are beneficial and well tolerated, including dimethylglycine (DMG) and a combination of pyridoxine (vitamin B6) and magnesium, both of which benefit roughly half of ASD cases. Vitamins A, B3, C, and folic acid; the minerals calcium and zinc; cod liver oil; and digestive enzymes, all offer benefit. Secretin, a triggering factor for digestion, is presently under investigation. Immune therapies (pentoxifyllin, intravenous immunoglobulin, transfer factor, and colostrum) benefit selected cases. Long-chain omega-3 fatty acids offer great promise. Current pharmaceuticals fail to benefit the primary symptoms and can have marked adverse effects. Individualized, in-depth clinical and laboratory assessments and integrative parent-physician-scientist cooperation are the keys to successful ASD management.

L13 ANSWER 19 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 9
ACCESSION NUMBER: 2002:767730 CAPLUS
DOCUMENT NUMBER: 137:288903
TITLE: Treatment of autism spectrum children with thiamine tetrahydrofurfuryl disulfide: a pilot study
AUTHOR(S): Lonsdale, Derrick; Shamberger, Raymond J.; Audhya, Tapan
CORPORATE SOURCE: Preventive Medicine Group, Westlake, OH, USA
SOURCE: Neuroendocrinology Letters (2002), 23(4), 303-308
CODEN: NLETDU; ISSN: 0172-780X

PUBLISHER: Neuroendocrinology Letters
DOCUMENT TYPE: Journal
LANGUAGE: English

AB In a pilot study, the clin. and biochem. effects of thiamin tetrahydrofurfuryl disulfide (TTFD) on autistic spectrum children were investigated. Ten children were studied. Diagnosis was confirmed through the use of form E2, a computer assessed symptom score. For practical reasons, TTFD was administered twice daily for two months in the form of rectal suppositories, each containing 50 mg of TTFD. Symptomatic responses were determined through the use of the computer assessed Autism Treatment Evaluation Checklist (ATEC) forms *. The erythrocyte transketolase (TKA) and thiamin pyrophosphate effect (TPPE), were measured at outset and on completion of the study to document intracellular thiamin deficiency. Urines from patients were examined at outset, after 30 days and after 60 days of treatment and the concns. of SH-reactive metals, total protein, sulfate, sulfite, thiosulfate and thiocyanate were determined The concns. of metals in hair were also determined

At the beginning of the study thiamin deficiency was observed in 3 out of the 10 patients. Out of 10 patients, 6 had initial urine samples containing arsenic in greater concentration than healthy controls. Traces of mercury were seen in urines from all of these autistic children. Following administration of TTFD an increase in cadmium was seen in 2 children and in lead in one child. Nickel was increased in the urine of one patient during treatment. Sulfur metabolites in urine did not differ from those measured in healthy children. Thiamine tetrahydrofurfuryl disulfide appears to have a beneficial clin. effect on some autistic children, since 8 of the 10 children improved clin. We obtained evidence of an association of this increasingly occurring disease with presence of urinary SH-reactive metals, arsenic in particular.

REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 20 OF 23 MEDLINE on STN DUPLICATE 10
ACCESSION NUMBER: 2002440331 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12197782
TITLE: Autism, an extreme challenge to integrative medicine. Part: 1: The knowledge base.
AUTHOR: Kidd Parris M
SOURCE: Alternative medicine review : a journal of clinical therapeutic, (2002 Aug) Vol. 7, No. 4, pp. 292-316. Ref: 114
Journal code: 9705340. ISSN: 1089-5159.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Consumer Health
ENTRY MONTH: 200210
ENTRY DATE: Entered STN: 29 Aug 2002
Last Updated on STN: 19 Oct 2002
Entered Medline: 18 Oct 2002

AB Autism, archetype of the autistic spectrum disorders (ASD), is a neurodevelopmental disorder characterized by socially aloof behavior and impairment of language and social interaction. Its prevalence has surged in recent years. Advanced functional brain imaging has confirmed pervasive neurologic involvement. Parent involvement in autism management has accelerated understanding and treatment. Often accompanied by epilepsy, cognitive deficits, or other neurologic impairment, autism manifests in the first three years of life and persists into adulthood. Its etiopathology is poorly defined but likely multifactorial with heritability playing a major role. Prenatal toxic exposures (teratogens) are consistent with autism spectrum

symptomatology. Frequent vaccinations with live virus and toxic mercurial content (thimerosal) are a plausible etiologic factor. Autistic children frequently have abnormalities of sulfoxidation and sulfation that compromise liver detoxification, which may contribute to the high body burden of xenobiotics frequently found. Frequent copper-zinc imbalance implies metallothionein impairment that could compound the negative impact of sulfur metabolism impairments on detoxification and on intestinal lining integrity. Intestinal hyperpermeability manifests in autistic children as dysbiosis, food intolerances, and exorphin (opioid) intoxication, most frequently from casein and gluten. Immune system abnormalities encompass derangement of antibody production, skewing of T cell subsets, aberrant cytokine profiles, and other impairments consistent with chronic inflammation and autoimmunity. Coagulation abnormalities have been reported. Part 2 of this review will attempt to consolidate progress in integrative management of autism, aimed at improving independence and lifespan for people with the disorder.

L13 ANSWER 21 OF 23 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2003:82298 CAPLUS
DOCUMENT NUMBER: 138:219855
TITLE: Vaccines, viruses, and voodoo
AUTHOR(S): Borchers, Andrea T.; Keen, Carl L.; Shoenfeld, Yehuda; Silva, Joseph, Jr.; Gershwin, M. Eric
CORPORATE SOURCE: Division of Rheumatology, Allergy and Clinical Immunology, University of California at Davis, Davis, CA, USA
SOURCE: Journal of Investigational Allergology and Clinical Immunology (2002), 12(3), 155-168
CODEN: JIAIEF; ISSN: 1018-9068
PUBLISHER: Hogrefe & Huber Publishers
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. Vaccinations are invaluable in protection from a wide variety of diseases that can cause substantial morbidity and mortality. Although a rare complication of vaccination, autoimmune disorders represent one of these morbidities. Recently, widespread public concern has arisen from case reports suggesting that similar to what has been observed after natural viral infections-there might be an association between specific immunizations and autoimmune diseases. Herein we address the biol. plausibility of such a connection, focusing particularly on the examples of hepatitis B, rubella, and measles-mumps-rubella (MMR) vaccinations, and the autoimmune diseases they are potentially associated with. Our review of the available data suggests that, for the general population, the risk:benefit ratio is overwhelmingly in favor of vaccinations. However, the possibility cannot be ruled out that, in genetically susceptible individuals, vaccination can result in the unmasking of an autoimmune disease triggered by the immunization. We also critically examine the existing data suggesting a link between immunization against MMR and autism, and briefly discuss the controversial evidence pointing to a possible relationship between mercury exposure from vaccines and autistic disorders. There is a continued urgent need for rigorously designed and executed studies addressing these potential assocns., although the use of vaccinations remains a critical public health tool for protection against infectious disease.
REFERENCE COUNT: 119 THERE ARE 119 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 22 OF 23 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2002293114 EMBASE
TITLE: The neuropathogenesis of mercury toxicity.
AUTHOR: Aschner M.; Walker S.J.

CORPORATE SOURCE: Dr. M. Aschner, Department of Physiology, Wake Forest Univ. School of Medicine, Medical Center Blvd, Winston-Salem, NC 27157-1083, United States. maschner@wfubmc.edu
SOURCE: Molecular Psychiatry, (2002) Vol. 7, No. SUPPL. 2, pp. S40-S41. .
Refs: 11
ISSN: 1359-4184 CODEN: MOPSFQ
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 007 Pediatrics and Pediatric Surgery
008 Neurology and Neurosurgery
032 Psychiatry
046 Environmental Health and Pollution Control
052 Toxicology
LANGUAGE: English
ENTRY DATE: Entered STN: 5 Sep 2002
Last Updated on STN: 5 Sep 2002
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L13 ANSWER 23 OF 23 EMBASE 
ACCESSION NUMBER: 2000396600 EMBASE
TITLE: Chromium, selenium, copper and other trace minerals in health and reproduction.
AUTHOR: Tuormaa T.E.
CORPORATE SOURCE: T.E. Tuormaa, 1.36 Castle St., Nether Stowey, Bridgewater, Somerset TA5 1LW, United Kingdom
SOURCE: Journal of Orthomolecular Medicine, (2000) Vol. 15, No. 3, pp. 145-156. .
Refs: 110
ISSN: 0317-0209 CODEN: JORMEI
COUNTRY: Canada
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 010 Obstetrics and Gynecology
016 Cancer
017 Public Health, Social Medicine and Epidemiology
029 Clinical Biochemistry
052 Toxicology
LANGUAGE: English
ENTRY DATE: Entered STN: 13 Dec 2000
Last Updated on STN: 13 Dec 2000
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER